

**THE EFFECT OF RAPID MAXILLARY EXPANSION
ON NASAL AIRWAY RESISTANCE, CRANIOFACIAL
MORPHOLOGY AND HEAD POSTURE**

James Peter McDonald

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A B S T R A C T

The adequacy of the nasopharyngeal airway has been found to be related to craniofacial development in that obstruction of the airway by excess adenoidal tissue, nasal septal deformity or other abnormal morphology in the area is associated with characteristic changes in craniofacial morphology.

The present study investigated the differences in nasal airway resistance, (to include the laminar and turbulent components), craniofacial morphology and head posture between a control group and an anomaly group exhibiting full transverse cusp bilateral crossbites and further investigated the changes that occurred in these parameters when the anomaly group was treated using rapid maxillary expansion.

The method error of both the measurement system for cephalometry and nasal airway resistance was tested by duplicate determinations and subsequent statistical analysis, as was the method error of the operator. All the variables in the study were found to be reproducible without systematic error and with a very small method error.

The results of the rhinomanometric readings showed that there were significant differences in nasal airway resistance between the control and anomaly samples, and that significant changes in nasal airway resistance occurred in the anomaly subjects when they were treated with rapid maxillary expansion. However when head posture was investigated, no significant differences in craniocervical angulation were discovered between the control and anomaly samples, or between the anomaly sample before and after treatment.

The craniofacial morphology values, measured using lateral cephalometric radiographs and postero-anterior radiographs, were found to exhibit some significant differences between the control and anomaly subjects, and to exhibit some significant changes where the anomaly sample was treated with rapid maxillary expansion.

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DECLARATION

This thesis is the original work of the author,
with the exception of the help and guidance
acknowledged in the text.

J P McDonald

CHAPTER 1

LITERATURE REVIEW

1.1 RHINOMANOMETRY

Rhinomanometry is the measurement of the pressure encountered by air passing through the nasal cavity (Clement 1984). It was introduced by Brown and Claesen in 1877, with an indirect procedure described by Zwaardemaker in 1889 whereby a plain mirror was held underneath the nostrils during expiration, the moisture in the exhaled breath being condensed onto its surface. The size of the patches and the time they took to disappear constituted a measure of the nasal passage. Kayser (1895) suggested a direct method for evaluating the nasal passage based on determination of:

1. the amount of air passing through the nose
2. the pressure in the nose during respiration and
3. the velocity of flow

He pointed out that if any two of these factors are known it is possible to calculate the third. Many methods have been published for the recording and measurement of nasal respiratory resistance including those of Aschan et al (1958); Rasmus and Jacobs (1969); Maran et al (1971); Kern (1973); MacKay (1979); Masing et al (1974); Broms et al (1982); Mygind (1980); Solow and Greve (1980); Gurley and Vig (1982).

The accurate recording of nasal respiratory resistance (NRR) is obviously important if the technique is to be used as a diagnostic and research tool. In addition standardisation needs to be agreed upon so that values gained can be meaningfully compared. Kern (1973, 1981) and Broms et al (1982) proposed some elements of standardisation, but it was not until the 8th Congress of the

European Rhinologic Society in 1980 in Bologna, Italy, and more importantly the International Meeting on Standardisation of Rhinomanometry in Brussels in 1983 that important recommendations were made concerning methods of measurement, terminology, calibration and the elaboration of results (Clement 1984). As far as results were concerned, it was agreed that

- a. preference should be given to the expression of the resistance at a fixed pressure rather than at a fixed flow, the reference pressure being 150 pascal.
- b. the equation $R = \frac{\Delta p}{\dot{V}}$ was accepted where R = resistance, Δp = the pressure difference and \dot{V} = the airflow.
- c. it was not necessary to standardise decongestion techniques and that each Rhinomanometrist should utilise the method of his own choice (normally nasal spray or exercise).
- d. for reliable measurements a minimum of 3-5 breaths should be recorded.

Stoksted (1951) utilised rhinomanometry in his work on pre- and post-surgical adenoidectomies of schoolchildren with adenoid problems and described its wider use as a diagnostic tool (Stoksted 1959). He further described the cyclic changes in the nasal airway, a phenomenon first noted in 1895 by the German rhinologist Kayser who described a consistent pattern of congestion and decongestion of the nasal mucosa which he termed the nasal cycle. In further studies Hasegawa and Kern (1977) performed experiments which showed 72% of the subjects demonstrated a clearly defined nasal cycle, with a resistance difference of 20% between the two sides right and left reversing or changing sides at least once for two consecutive calculations

during the seven hour testing time. The authors further suggested that normal individuals are not usually aware of this phenomenon because the total nasal resistance usually remains fairly constant and is in fact less than the resistance of either one of the individual nasal passages. In the present study 0.1% xylomethazoline hydrochloride as a nasal spray was administered to the subject one half hour before each recording session to eliminate the cyclic turbinate engorgement (Lenz et al 1985) and any nasal congestion associated with infection or allergy, hence establishing a free airway (Henriksen and Wenzel 1984).

Stoksted (1959) suggested that the resistance in the nose was determined by the two factors, one being the size of the internal orifice and the other the erectile or cavernous tissue of the turbinates. He pointed out that on each inspiration the nasal wings moved outwards dilating the internal orifice and reducing the pressure during the inspiration phase, a movement controlled by the levator labii alaeque nasi muscle (Van Dishoeck 1942). Opinions vary however as to the location of the nasal valve. Mink (1920) applied the term to the main site of nasal resistance placing it at the junction of the upper and lower lateral alar cartilages.

Proctor (1977) located the valve to the region lying between the junction of the upper and lower lateral cartilages and the pyriform aperture. Further studies by Haight and Cole (1983) showed that the main air flow resistance is normally confined to a short nasal segment of a few millimetres situated in close proximity to the junction of the cartilaginous vestibules and the rigid bony cavity of the nose.

Aschen et al(1958) used a small mask placed over the nose in order to measure anterior nasal resistance, calculating it by utilising pressure flow equation $NRR = \frac{\Delta p}{\dot{V}}$. These important experiments form the basis of present methodology, even though the suggestion that nasal resistance could be measured in a single nostril by obstructing

the other with a cotton wool roll is questionable given that each anterior measurement is independent of the pharyngeal component. The technique however proposed by Ashen et al (1958) was used by Linder-Aronson (1970) in a study to measure nasal airflow in subjects with enlarged adenoids, before and after adenoidectomy. He demonstrated that nasal airflow at a fixed pressure was low in children with enlarged adenoids, high in those with small adenoids, and that airflow increased and significant changes occurred in the dentition after adenoidectomy in children who had been mouth breathers pre-operatively because of nasal obstruction. The size of the nasopharyngeal cavity also changed in children who switched from mouth to nose breathing after adenoidectomy.

Inglestedt et al (1969) developed the technique of Ashen and co-workers to measure total or bilateral resistance measurements, but since nasal resistance increased with flow rate the measurements were made at a fixed flow rate to facilitate comparisons. Fischer (1970) proposed a technique for measurement of nasal resistance of both right and left nasal cavities. He measured pressure differences at the one nostril while measuring a flow in the other hence enabling more accurate calculations of resistance of right and left components, greatly improving the diagnostic value of the rhinomanometer.

Kortekangas (1972) used the same technique to compare the differences between values obtained by rhinomanometry for anterior (unilateral) and posterior (bilateral) measurements. Bachman (1973, 1976, 1984) proposed a threshold value utilising a fixed pressure, later agreed at 150 pascal.

A modification of the above techniques for anterior measurements has been used in this study together with the advantages of recent computer technology (Solow and Sandham 1989). These researchers further measured the values for laminar (K1) and turbulent (K2) components of the airflow (Rohrer 1925).

Courtiss and Goldwyn (1983) attempted to measure the effects of nasal surgery on the laminar/turbulent flow pattern but came to the conclusion that rhinomanometry was not a reliable method of doing so. Subsequently Solow and Sandham (1991) tested a sample of 20 dental students with no history of nasal airway obstruction with a view to describing the relative contributions of laminar and turbulent flow to the total pressure drop across the nose, utilising a Rohrer programme. (See Table 1). They discovered that in unilateral breathing there was a predominance of turbulent flow but on testing the bilateral breathing a switching of airflow characteristics occurred with a marked reduction in the turbulent components of flow. The authors suggested that the Rohrer equation coefficients might provide information about nasal airflow which are physiologically more appropriate than conventional resistance calculations, which are based on a single point on the rhinomanometric pressure flow curve.

Nasal resistance in normal children has been studied by Solow and Greve (1980), Saito and Nishihata (1981), and Principato and Wolf (1985), hence establishing norm values against which the nasal resistance values of the anomaly sample of the present study can be compared. These are summarised below in Table 2.

TABLE 1

NASAL AIRFLOW CHARACTERISTICS IN A NORMAL SAMPLE

Study	Subjects	Age Range	Results
Solow and Sandham (1991)	20 (8 male, 12 female)	22.9 years to 27.8 years	Unilateral NAR 300 Pas/L/S Bilateral NAR 250 Pas/L/S Unilateral K1-113 K2-807 Bilateral K1-147 K2-348

TABLE 2

NORMAL VALUES FOR NASAL AIRWAY RESISTANCE

Study	Subjects	Age Range	Mean NAR	Significance
Principato & Wolf (1985)	498 (236 female, 262 male)	4 to 16 years	CmH ₂ O/L/sec 4 yrs - 8.28 ↓ 16 yrs - 3.18 (Ant only)	NAR varies inversely with age - highly significant. 95% confidence level
Solow & Greve (1980)	17 (11 female, 6 male)	8 to 14 years	CmH ₂ O/L/sec Post - 0.15 Ant (R) - 1.19 Ant (L) - 2.96	Mean differences not significant at 5% level
Saito & Nishihata (1981)	397 (212 female, 185 male)	5 to 17 years	At flow rate 3 l per minute Ant NAR 700 pas/L/sec at age 5-8 300 pas/L/sec at age 16	N/S

1.2 HEAD POSTURE, CRANIOFACIAL MORPHOLOGY AND NASAL AIRFLOW

As early as the 1860s anthropologists realised that for cephalometric studies skulls had to be orientated in a similar way to the natural head position in the living, to make any comparison valid. Von Baer and Wagner (1861) and Broca (1862) decided that a horizontal or vertical reference line outside the cranium should be used, preference being given generally to the horizontal. In 1884 the Frankfort horizontal plane was adopted at the Craniometrical Conference in Frankfurt.

With the advent of cephalometric radiography in the 1930s this plane was utilised as the basis for many analyses. As research utilising cephalometric analysis developed it became important for a standardised natural head position to be used, and in 1957, Bjerin utilised photographs of the facial profile tracings of the enlarged photographs on the cephalometric tracing. Linder-Aronson in 1979 placed a horizontal pencil mark on the cheek of a patient standing in a relaxed position outside the cephalometer in front of a mirror. Once inside the cephalometer the head was positioned with the pencil mark and a projected horizontal light beam superimposed. Vig et al (1980) taped a device containing a radio-opaque fluid level to the subject's head after obtaining the self balance position. Once inside the cephalometer the meniscus was paralleled to a wire. However these methods do not take into account cervical column position.

Solow and Tallgren (1971_{a,b}) utilised a method based on a natural body posture, followed by a procedure in which the patient finds the natural head position by utilising a mirror to enable him to look straight into his own eyes. Using this technique it is possible to measure the position of the head in relation to the true vertical, as well as in relation to the cervical column. Siersback-Nielson and Solow (1982) successfully

tested this method for reproducibility as did Sandham (1988). Cook (1990) undertook a five year longitudinal study of the stability of natural head posture, and found that after an initial deterioration over the first year, the variance of NHP (9.24°) remained significantly less than the variance of intracranial reference points to the vertical (25° to 36°).

The availability of a reliable and reproducible method of measuring natural head and cervical column position is a prerequisite for research into the complex interaction between head posture, craniofacial morphology and airway adequacy. Schwartz (1926) suggested a relationship between head posture and craniofacial morphology when he attributed the development of a Class II malocclusion to hyperextension of the head relative to the cervical column during sleep. Gresham and Smithells (1954) supported this hypothesis; they noted a larger prevalence of Class II malocclusion in subjects with “poor neck posture” as did Bjork (1955). In 1977, Solow and Tallgren in a detailed study of head posture and craniofacial morphology, noted that the craniocervical angulation showed a higher correlation with morphology than the position of the head in relation to the true vertical. Extension of the head in relation to the cervical column was found to be associated with a large anterior and a small posterior facial height, small anteroposterior craniofacial dimensions, large inclination of the mandible to the anterior cranial base and to the nasal plane, facial retrognathism, a large cranial base angle and a small nasopharyngeal space. In contrast, flexion of the head showed exactly the opposite. These findings were similar to those made by Opdebeek et al (1978) and Marcotte (1981). With regard to the relationship between airway adequacy and type of malocclusion, Watson et al (1968) found no association between airway adequacy and type of malocclusion or craniofacial morphology. Linder-Aronson (1970) demonstrated that patients with upper airway obstruction due to enlarged adenoids had a significantly different craniofacial morphology than that of a control group. The

effect on the dental structures included retroclination of the lower incisors and reduction of the overjet, with a narrow upper arch between the first molars often resulting in a crossbite. These patients were obligate mouth breathers (Schweiger 1966).

After adenoidectomy, Linder-Aronson (1974,1975) found that the differences in morphology originally seen between the sample and the control group decreased significantly, this indicated a direct relationship between craniofacial morphology and adenoidal obstruction. Linder-Aronson suggested that in mouth breathing the tongue position is lowered and when adenoids are removed the resting tongue position may be raised resulting in reversal of the anomaly trends. The relationship between craniofacial morphology and rhinomanometrically measured airway adequacy in adenoid subjects has been subsequently shown by Bushey (1977), and the relationship between craniofacial morphology and radiographically measured nasopharyngeal airway adequacy has been demonstrated by Sosa et al (1982). As well as research into the effect of adenoids on the airway, other airway anomalies have been studied. Respiratory obstruction has been shown in patients with cleft lip and palate by Drettner (1960), Warren et al (1969) and Sandham and Solow (1988). Particularly pertinent to the present study was the improved nasal airflow in orthodontic patients after rapid maxillary expansion as demonstrated by Hershey et al (1976) and Loreille and Bery (1981).

In order to explain the similarity between the craniofacial morphology of those subjects with a large craniocervical angulation and those with obstructive airway due to enlarged adenoids, Solow and Kreiborg (1977) suggested an hypothesis to account for the association between head posture, craniofacial morphology and airway obstruction; this has been termed the control hypothesis. They suggested a circular chain of interactions involving

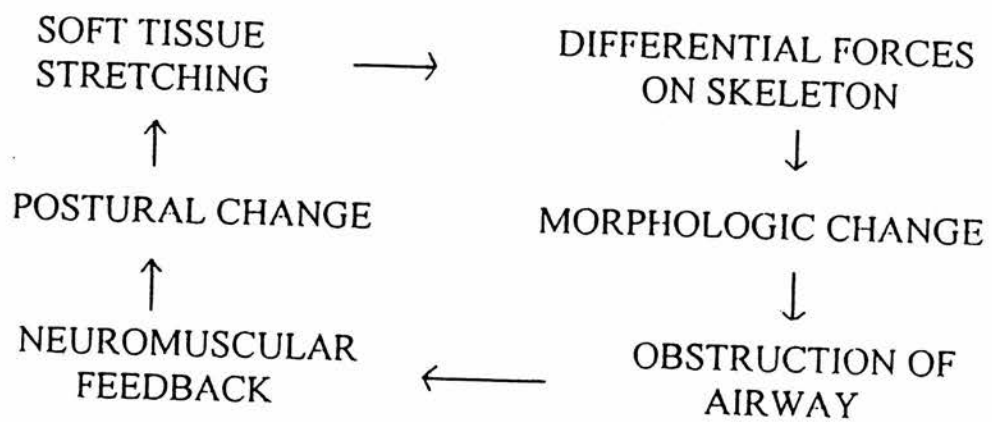


Figure 1
Solow and Kreiborg (1977)

- a. a change in airway adequacy
- b. neuromuscular feedback
- c. a change in craniocervical angulation
- d. passive stretching of the soft tissue layer of the face and neck
- e. morphologic change resulting in "adenoidal facies". (see Figs 1 and 2).

In 1979 Solow and Greve demonstrated the association between craniocervical angulation and nasal airway resistance in a group of children before and after adenoidectomy. The findings demonstrated that a large craniocervical angulation was seen in conjunction with a large NAR in the subjects before adenoidectomy, the NAR and craniocervical angulation reducing significantly after the operation, there being a reduction of 2° in craniocervical angulation and in the position of the head in relation to the true vertical some two months after adenoidectomy. Woodside and Linder-Aronson (1979) performed similar studies with similar results, as had Ricketts (1968).

The relationship between head posture and airway adequacy was also demonstrated experimentally by Vig et al (1980). These findings confirmed the control hypothesis.

Solow et al (1984) examined three sets of associations in a non-pathological group of subjects with no history of airway obstruction. The aim of the study was to examine the predicted association between head posture, craniofacial morphology and airway adequacy. Moderate correlations were discovered, enough to indicate the presence of a general control mechanism in craniofacial development. Given the relationship between airway adequacy and craniofacial morphology and between airway adequacy and craniocervical posture demonstrated by Solow et al (1984), RME would be expected to cause changes both in craniocervical posture and conceivably in craniofacial morphology. This has not been investigated to date.

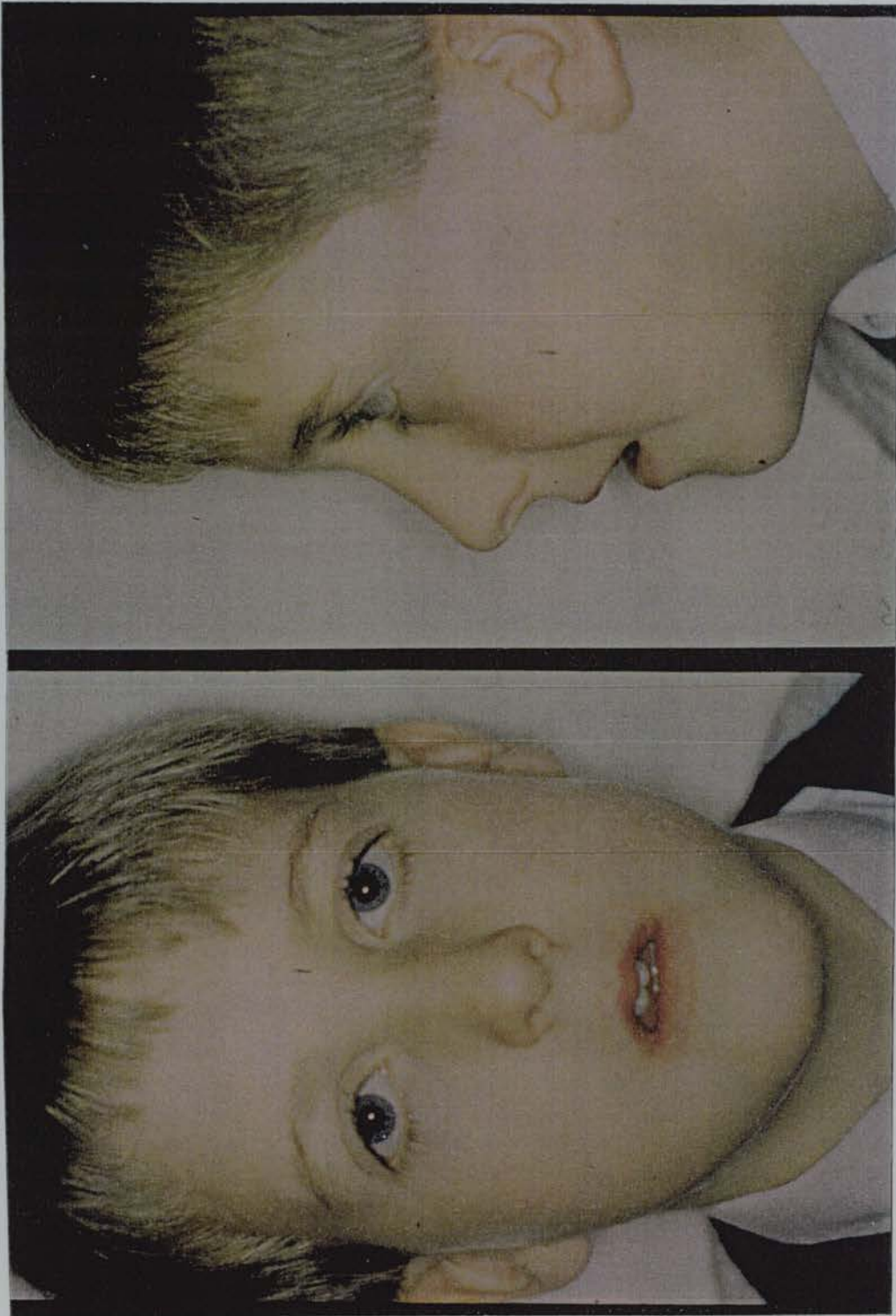


Fig. 2 Typical facial characteristics in a child from the anomaly sample

1.3 RAPID MAXILLARY EXPANSION

History

Rapid maxillary expansion is not a new technique, the first published work appearing in 1860 when E H Angell described the rapid expansion of the upper arch to provide space for maxillary canines. The main supporters of the technique at that time however were not Orthodontists - the most vociferous supporter being Brown (1909), a noted Otorhinologist who published in the Dental Cosmos supporting claims that rapid maxillary expansion allowed the straightening of a deviated nasal septum and provided relief of hypertrophied conditions of nasal and pharyngeal mucous membranes. Pfaf (1905) was of the opinion that routine orthodontic expansion of the dental arch lowered the palatal vault and induced straightening of the nasal septum, in turn moving the septum away from the turbinate bones and permitting an increased air volume. In the early 1900s many articles appeared pointing to the inter-relations of certain orthodontic and rhinological treatments and emphasising that rapid maxillary expansion in particular had consequences far beyond the teeth and mouth.

Since that time, the procedure has gone through periods of popularity and decline, particularly in comparison to slower methods of expansion. A review of the contemporary literature indicates that there were numerous disagreements as to the indications and efficacy of rapid maxillary expansion. Angle (1907) supported slow maxillary expansion whilst C H Hawley (1912) and H A Pullen (1912) opted for RME.

Aetiology

The aetiology of lateral discrepancies resulting in either unilateral or bilateral posterior crossbites can be either general or environmental. Harvold et al (1973) in their experiments on the development of dental malocclusions were able to create narrow maxillary dental arches in rhesus monkeys by converting them from nasal to obligatory oral respiration. This environmental approach was supported by Graver et al (1975).

Histology

Sutures - As the midpalatal suture is of paramount importance in any study involving rapid maxillary expansion, close examination of its composition and characteristics both before and after rapid expansion is essential.

Development of a normal suture without RME - Melsen (1975) traced the development of the suture from birth to adulthood by histologic means utilising material from human cadavers. She found that in infancy the suture, looked at in a vertical coronal section, is traced the development of the suture from birth to adulthood by histologic means utilising material from human cadavers. She found that in infancy the suture, looked at in a vertical coronal section, is Y-shaped binding the vomer with the palatine processes. As the suture ages the junction between the three bones assumes more of a T-shape and by adolescence the sutural course may become so interdigitated that jigsaw-like interlocking may take place. Melsen stresses that this latter developmental characteristic is unique to humans, so the results of animal experiments cannot be directly transferred to man. Melsen (1972) however working on human biopsy material obtained by a trepanning technique revealed much in common with animal findings up to the period of maximal pubertal growth.

Human autopsy material was also used by Persson and Thilander (1977) to examine the timing and ossification rates. They found the earliest closure to be in a 15 year old female whilst the oldest unossified suture was in a 27 year old

female, a greater degree of obliteration occurring posteriorly rather than anteriorly.

The fact that the suture starts to ossify posteriorly (Davida 1926) is important when planning surgical intervention in sutural division.

Suture after RME - histologic studies of the suture opening by Derichsweiler (1953) and Cleall et al (1979) have demonstrated rapid cellular adjustments at the intermaxillary suture, new bone being formed in the palatal void. Ekstrom et al (1977) found that the mineral content within the suture rose rapidly during the first month after the completion of suture opening, the mineral content of the adjacent bone decreasing sharply through the first month but returning to its initial level within three months. Ten Cate et al (1977) found that the separation of the suture involved tissue injury which was followed by repair and regeneration of the suture.

Brin et al (1981) investigated the effects of age on sutural cyclic nucleotides, and came to the conclusion that the bone cells of older animals are less responsive to tensile forces than the corresponding cells in younger animals hence suggesting that the reasons for differences in clinical response between various age groups may result from variations in cellular biology.

Techniques

Removable expansion plates are not effective if significant skeletal changes are required, even though limited midpalatal separation has been recorded (Krebs 1964).

The use of removable appliances can only be justified in the deciduous or early mixed dentition. The main problem, as indeed with some of the fixed palatal expanders, is the lack of rigidity (the resistance to rotation). Nearly parallel opening by a rigidly fixed appliance is required, or the dento-alveolar components will tilt buccally diminishing the amount of basal bone expansion that is necessary. Chaconas (1975) showed that the appliance form determines the

shape of the expansion, further demonstrating the ineffectiveness of the removable expander.

Angell (1860) recommended a fixed expansion appliance utilising a double jack screw with opposing threads, and succeeded in widening the maxillary arch of a 14 year old girl by $\frac{1}{4}$ " in two weeks. Since then, appliances cemented to the teeth either in the form of bands or cap splints have been the vehicles of choice. Again, rigidity is of prime importance so the quadhelix (Ricketts 1957) which is attached only to the maxillary first molars can only be effective in cleft palate cases. The Hyrax, Haas, Derichsweiler or Biederman appliances, which are attached to both first permanent molars and the first permanent premolars certainly have the rigidity to separate the palatal shelves and are widely used. However Barbar and Sims (1981), Langford and Sims (1982) and Odenrich et al (1982) all recorded marked buccal root resorption in first permanent premolars when these were used as buttresses for the Biederman rapid maxillary expanding appliances. These defects tended to gradually repair when the pressure was relieved.

Silver/copper alloy cast cap splints, as described by Grossman (1963), have a number of advantages over band retained appliances. Cast splints, which are extremely rigid, spread the load levels over the whole buccal tooth bearing area and also relieve intercuspal locking whilst the expansion is taking place. The disadvantage of poorer oral hygiene can be minimised by irrigation beneath the palatal acrylic surface of the appliance using a water syringe supplied to the patient.

The screws used in rapid maxillary expander appliances are normally Hyrax (Dentaurum 602-813), Glenross VI or Leone 620 all of which give between 11 and 18mm of expansion. Isaacson et al (1964) however utilised a springloaded screw called a Minne Expander (Minnesota Dental School). More recently bonded full coverage appliances had been described (Mondro 1977, Howe 1982 and Spolyar 1984).

Forces and Relapse

Rapid maxillary expansion takes place when the lateral force applied to the teeth and maxillary alveolar processes exceeds the limits needed for orthodontic tooth movement. This pressure then acts as an orthopaedic force, compressing the periodontal ligament, deforming the alveolar processes and opening the midpalatal suture.

The forces generated by rapid palatal expanding appliances are high. These forces have been investigated by Isaacs (1964) who used a strain gauge to study the forces produced. Isaacs found there may be up to 20lb of force applied during suture opening and it was his view that the heavy forces in suture opening provided minimal tooth movement and maximum reposition of the maxillary segments. He also believed that in order to prevent relapse of the bone segments these heavy forces must be allowed to dissipate throughout the whole maxillary complex before any appliances are removed. With such heavy forces it is reasonable to assume that the effects of the procedure spread widely into the bones of the facial complex.

Mesnard (1929) reported the use of fixed appliances for separation of the maxilla and described some of the accompanying changes. In addition to dislocation of the maxilla in the midline, he documented the lowering of the vault of the palate and floor of the nose, the straightening of the nasal septum and the restoring of nasal permeability. An examination of occlusal films by Wertz (1970) showed that the opening of the midpalatal suture extends through the horizontal plates of the palatine bones but only to a small degree. Kudlick (1973) working on a human dried skull came to the conclusion that it was the sphenoid bone not the zygomatic arch that was the main buttress against maxillary expansion. He emphasised that the pterygoid plates of the sphenoid, although bilaterally positioned, do not have a mid-sagittal suture that allows them to be displaced laterally. This limiting effect of the pterygoid plates of the sphenoid

minimises dramatically the ability of the palatine bones to separate at the mid-sagittal plane. The pterygoid plates can only bend to a limited extent as pressure is applied to them, and their resistance to bend increases dramatically in the parts closer to the cranial base where the plates are much more rigid (Timms 1980).

As far as the separation of the maxillary segments is concerned, the maxillary suture was found to separate in a pyramidal shape, the base of the pyramid located at the oral site of the bone (Haas 1961, 1970). The relation between the amount of sutural separation and the amount of molar expansion was studied by Krebs (1964), who placed implants in the alveolar processes lingual to the upper canines and along the infra-zygomatic ridge, buccal to the first upper molars. He found that the amount of sutural opening was equal to or less than one half of the amount of dental arch expansion. He further found that the sutural opening was on average more than twice as large between the incisors as between the molars. He also found that although dental arch width was maintained during fixed retention, the distance between implants in the infra-zygomatic ridges decreased through the first three months of fixed retention by an average of 10-15%, this relapse continuing during retention with removable appliances so that after an average period of fifteen months only 70% of the infra-zygomatic maxillary width increased was maintained.

The final position of the maxilla was investigated by Haas (1961) and Wertz (1970). They both found it to be frequently displaced downward and forward but the final position was unpredictable due to an element of relapse that could be partial or complete. In addition the maxillae were found to tip relative to one another around a fulcrum approximately at the fronto-maxillary suture. This would explain the increased width gain at the dental rather than the sutural level.

One of the most obvious signs accompanying the opening of the midpalatal suture is the opening of a diastema between the maxillary central incisors, an event about which both the parents and the patient must be prewarned. The incisors separate approximately one half the distance of the opening of the expansion screw (Haas 1961) but the amount of separation between the central incisors cannot be reliably used as an indicator of the sutural opening. After this separation, the incisor crowns converge due to the force from the stretched trans-septal fibres. Once the crowns contact, the fibres continue to pull giving a tendency for the maxillary central incisors to be extruded and become more upright or tip lingually (Wertz 1964), thus shortening the arch length. This latter movement can be forestalled by employing sectional fixed appliances utilising tubes soldered to the rapid maxillary expansion appliance, a technique developed for this present study. The maxillary posterior teeth have a tendency to tip buccally as force is applied.

Skeiller (1964) and Hicks (1978) suggest that slower maxillary expansion is less prone to relapse because the lower force levels cause less alveolar plate warpage and less disruption of the adjacent bones. Wertz (1970) found that rapid maxillary expansion led to an increase in width of the maxilla due to a lateral rotation of each half, this rotation tending to return to normal after termination of active expansion. Timms (1976) re-examined twenty six patients five years out of retention; a mean relapse of intermolar width of 4mm was observed giving a mean residual expansion of 44%.

Wertz and Dreskin (1977) stated that maxillary skeletal width can be expanded with no relapse in younger patients, older patients having a significant tendency to relapse.

Linder-Aronson and Lindgren (1979) re-examined twenty three patients and found that the final increase in width between the upper first molars five years post-retention was 45% of that initially achieved by rapid maxillary expansion could be found. The role of

the palatal muco-periosteum in relapse was investigated by Cotton (1978) who suggested that relapse between the maxillary first molars may be related to the stretched fibres of the attached palatal mucosa. Maguerza and Shapiro (1980) attempted to relieve the stretch of the muco-periosteum after expansion by making incisions along the palate down to the cortical bone, some 3mm away from the teeth. However the incisions did not effectively reduce the amount of relapse.

As to the length of retention time, providing this is in excess of the three months suggested by Ekstrom et al (1977), there would appear only to be a weak association between the residual expansion and length of retention.

Greenbaum and Zachrisson (1982) examined the effect of palatal expansion therapy on the periodontal tissues by comparing patients with orthodontic treatment alone, rapid maxillary expanding appliances, and slow (quadhelix) palatal expanders. They found that the differences among the groups were not significant and clinically of small magnitude.

Secondary effects of RME on Mandibular Arch Width

The effects of rapid maxillary expansion on the mandibular arch width appear to be unpredictable, with Gryson (1977) recording no significant changes in the trans-canine and trans-molar mandibular widths whereas Sandstrom et al (1988) discovered statistically significant expansion of the inter-canine and inter-molar widths. Wertz (1977) observed mandibular intermolar width increased at the completion of maxillary suture opening and Haas (1965, 1970) recorded that the mandibular teeth became uprighted as the maxillary teeth expanded.

Anteroposterior orthopaedic forces

Immediately after the palate has been split, with concomitant breakage of adjacent sutures, it would seem to be an attractive proposition to apply orthopaedic forces to the maxillae, in order to move them mesially or distally. Haas (1961) applied forces to the maxilla following RME and reported an enhanced response. McNamara (1987) and Wemmer (1988) used Delaire face masks attached to the rapid palatal expansion appliance to bring post-normal maxillas forward immediately after rapid maxillary expansion. It is generally accepted that the midpalatal suture ossifies in the mid teens, after which rapid maxillary expansion becomes more problematical and unpredictable.

Surgical aspects

Different surgical approaches can be used to help correct maxillary constrictions with or without using a rapid maxillary expansion appliance. Surgery is particularly effective in overcoming the strong resistance of the maxillary complex after growth has completed. The surgical palatal expansion can be accomplished by surgically moving the maxilla with lateral corticotomies (Lines 1975), by surgically undermining the maxillae to facilitate expansion using an RME appliance, (Glassman 1984 and Alpern and Yurosko 1987) or by surgical separation of the palatal shelves (Bell and Epker 1976 and Kraut 1984). Mossaz et al (1992) have described a technique of unilateral and bilateral corticotomies for correction of maxillary transverse discrepancies.

It is worth remembering however that surgical procedures to expand the palate without rapid maxillary expansion run the risk of severe stretching of the palatal mucoperiosteum, with consequent tendency towards relapse.

Current best practice for these patients who have a fused midpalatal suture is to employ surgery to separate the suture followed by semi-rapid (one turn per day) expansion with a cast splint appliance.

1.4 RAPID MAXILLARY EXPANSION AND NASAL RESPIRATORY RESISTANCE

The two key questions that need to be asked when investigating the changes in nasal respiratory resistance associated with RME are:

1. Is there a change in NRR - and if so is it of clinical significance?
2. If the change is of clinical significance how permanent is it?

There can be no doubt that anatomically there is an increase in the width of the nasal cavity immediately following expansion, particularly at the floor of the nose adjacent to the midpalatal suture (Haas 1961). Haas investigated a sample of 80 cases of which 8 were over 20 years old. His work was supported by Wertz (1970).

The maxillae separate, and the outer walls of the nasal cavity move laterally effecting an increase in intra-nasal capacity. Pavlin et al (1984) using laser holography, albeit on dried skulls, calculated the average nasal cavity width gain to be 1.9mm while Gray (1975) suggested that at the important level of the inferior turbinates, the width gain can be as much as 8mm. Montgomery, Vig et al(1979) in a computed tomography study found the effects of RME on the nasal cavity to be progressively less towards the back of the nasal cavity, a finding which correlates with histological examination.

Warren et al (1987) concerning the small increase in binasal width (average 0.5mm) pointed out that the airflow varies inversely as the fourth power of the radius of the tube through which it passes, hence a small increase in the radial dimensions of the tube means considerable increases in the flow.

Hershey et al (1976) investigated 17 subjects, 6 male and 11 female, all mouth breathers. The palates were expanded using Biederman appliances and retained for three months after completion of expansion. They reported a reduction of nasal respiratory resistance by an average of 45% after rapid maxillary expansion, and

this was stable for one year after appliance removal. These findings were supported by Turbyfill (1976) who found in his study an average decrease of 53% in nasal respiratory resistance after rapid maxillary expansion. Graber (1975) believes that the improved nasal breathing apparent after RME is only a temporary measure, simply because children have more lymphoid tissue than adults and spontaneous resolution occurs through growth, automatically improving nasal breathing without orthodontic intervention. Wertz (1970) concluded that opening of the midpalatal suture for the purpose of increasing nasal permeability cannot be justified unless the obstruction is shown to be in the lower anterior part of the nasal cavity, and accompanied by a relatively severe maxillary arch width deficiency. Hargerink et al (1987,1989) concluded that due to the high variability of individual response, rapid maxillary expansion is not a predictable means of decreasing nasal respiratory resistance, and found no correlation between the amount of expansion and changes in nasal resistance. White and Woodside (1989) however found an average reduction in nasal airway resistance of 48.7%, which was statistically significant at the $p < 0.005$ level. This reduction also appeared stable throughout the post-treatment observation period (maximum one year), and was highly correlated to the initial degree of nasal resistance prior to rapid maxillary expansion. Those individuals with the greater initial resistance tended to have greater reductions in airway resistance following the expansion.

It can be seen from the conflicting results obtained by various workers that the inter-relationship between rapid maxillary expansion and nasal respiratory resistance remains unresolved. In part it may be due to the differing methods of obtaining rapid palatal expansion, and to methods of measuring nasal respiratory resistance. The aim of the present study is in part to continue research into the inter-relationship between RME and NRR, but in particular to measure any differences that occur in the balance between the laminar and turbulent airflow before and after RME.

1.5 SUMMARY

The adequacy of the nasopharyngeal airway has been found to be related to craniofacial development. Obstruction of the airway by adenoid tissue, nasal septal deviation or abnormal morphology of the area is associated with characteristic changes in craniofacial morphology such as long anterior face height, facial retrognathism and a steep inclination of the mandibular plane often with a high palate and crossbite (Linder-Aronson 1970 and Solow et al 1984).

Some studies have found the changes to be reversible after adenoidectomy which improves nasal airway patency (Linder-Aronson 1974,1975) and a control mechanism for facial growth has been proposed to account for the relationships between airway adequacy, craniofacial morphology and craniocervical postural relationships (Solow and Kreiborg 1977). It is therefore important to be able to accurately measure nasal airway resistance so that the effect of operative procedures in the area, such as rapid maxillary expansion (RME) can be determined.

Nasal airway resistance (NAR) is a measure of airway adequacy. It can be recorded by rhinomanometry, a non-invasive technique which measures the resistance to airflow by simultaneous recording of flow and the pressure drop over the nose (Aschan et al 1958, Massing 1965, Kern 1973, Solow and Greve 1980 and Bachmann 1982). Recent technical advances based on microcomputer technology have made recording easier, but most studies have used threshold values for pressure or flow to calculate nasal airway resistance (Sandham and Solow 1987). Proposals have been made concerning the more accurate determination of Laminar (K1) and Turbulent (K2) flow coefficients (Rhinomanometric Meeting Berlin 1987) using a mathematical model known as the Rohrer equation.

Equipment is now available to enable such recordings to be made. This provides more valuable and accurate data and serves as a basis for improved understanding of the effect of RME on nasal airway adequacy.

The recording by computer of linear and angular variables for craniofacial morphology (derived from co-ordinate data digitised from standardised cephalometric radiographs), is a procedure which has been developed, error tested and utilised in the Department of Preventive Dentistry, Edinburgh University, for a previous study (Sandham 1987) but based on an analysis of craniofacial form by Bjork, and computer programmes developed by Solow (1966). The recording of such variables relating to transverse relationships was digitised from postero-anterior radiographs using software developed specifically for the present study.

An association exists between craniocervical angulation and nasal airway resistance, a large craniocervical angulation being seen in conjunction with a large nasal airway resistance in a group of subjects before adenoidectomy. After the operative procedure, nasal airway resistance and craniocervical angulation was reduced (Linder-Aronson 1984). An alteration in craniocervical and possible cranio-hyoid relationships may therefore be detected in the present study if improvement in airway adequacy due to treatment with rapid maxillary expansion is found.

Previous studies have suggested that maxillary expansion may be justified on airway considerations alone but Warren et al (1987) and Hartgervink et al (1987) both suggest that the technique is not a predictable means of decreasing nasal airway resistance. However in the present study all the subjects in the anomaly group were treated by rapid maxillary expansion (RME) as part of a course of orthodontic treatment for bilateral crossbite.

2. AIMS AND OBJECTIVES OF THE STUDY

The review of the literature has shown that there is relatively little standardised information on Nasal Airway Resistance in children and on the effects of rapid maxillary expansion in those with full transverse cusp bilateral crossbite.

It was the aim of the present study therefore -

1. to compare craniofacial form, head posture and nasal airway resistance between a control sample and a sample of children with full transverse cusp bilateral crossbite and
2. to determine the effect on these parameters of rapid maxillary expansion on children in the anomaly sample

The aim of the study therefore is to investigate -

- a. craniofacial form
- b. head posture
- c. nasal airway resistance

in a group of children aged 10 to 15 years (inclusive) undergoing RME.

AIM 1

To compare the control and anomaly groups at the base line - Time 1 - for a, b and c above.

AIM 2

To determine the changes in the anomaly sample following treatment with RME - Time 2 - in terms of a, b and c above.

The Null hypothesis to be tested are:

AIM 1

- i. There will be no difference in the mean values of Nasal Respiratory Resistance, including the values for laminar and turbulent airflow, between the control and anomaly sample.
- ii. There will be no difference in the craniocervical angles NSL/OPT and NSL/VER between the control and the anomaly sample.
- iii. There will be no difference in the following lateral craniofacial morphology values between the control and anomaly sample

a. linear values:	n-s	sp-pm
	n-sp	ss-pm
	n-gn	pgn-cd
	s-ba	oj
	s-pm	ob
	sp-gn	
b. angular values:	n-s-ba	ss-n-pg
	pm-s-ba	NSL/NL
	s-n-sp	NSL/ML
	s-n-ss	NL/ML
	s-n-sm	NSL/MBL
	s-n-pg	ML/RL
	ss-n-sm	
c. airway dimensions:	pm-ad ₁	
	pm-ad ₂	
	pm-ad ₃	
	tu-ad ₃	

- iv. that there will be no difference in the transverse craniofacial dimensions between the control and anomaly sample:

CNR-CNM	MIR-MIL
CNM-CNL	MIR-C ₂
CNR-CNL	C ₂ -MIL
EMR-EML	NASDIM
UMR-UML	MAXDIM
LMR-LML	

AIM 2

- i. there will be no change in the mean values of Nasal Respiratory Resistance, including the values for laminar and turbulent airflow, before and after RME.
- ii. there will be no difference in the craniocervical angles NSL/OPT and NSL/VER before and after RME.
- iii. there will be no change in the following lateral craniofacial morphology values before and after RME.

a. linear values:	n-s	sp-pm
	n-sp	ss-pm
	n-gn	pgn-cd
	s-ba	oj
	s-pm	ob
	sp-gn	

b. angular values:	n-s-ba	ss-n-pg
	pm-s-ba	NSL/NL
	s-n-sp	NSL/ML
	s-n-ss	NL/ML
	s-n-sm	NSL/MBL
	s-n-pg	ML/RL
	ss-n-sm	

c. airway dimensions:	pm-ad ₁
	pm-ad ₂
	pm-ad ₃
	tu-ad ₃

iv. that there will be no change in the transverse craniofacial dimensions before and after RME

CNR-CNM	MIR-MIL
CNM-CNL	MIR-C ₂
CNR-CNL	C ₂ -MIL
EMR-EML	NASDIM
UMR-UML	MAXDIM
LMR-LML	

3. CLINICAL MATERIAL AND METHODS

3.1. Clinical Subjects

- a. The anomaly study group comprised 72 children, 41 female and 31 male, all within the age group 10-15 years inclusive (see Fig 3). The mean age of the females was 12 years 8 months, and of the males 12 years 9 months (see Table 3). They were all patients that had been referred to Victoria Hospital, or to Edinburgh Dental Hospital by General Dental Practitioners, General Medical Practitioners or Consultant ENT colleagues.

The criteria for inclusion in the study were that the subjects exhibited a full transverse cusp bilateral crossbite, and had not undergone any previous tonsillar, nasal or adenoidal surgery.

- b. The control sample comprised 36 children, 24 female and 12 male, again all within the age group 10-15 years inclusive (see Fig 4). The mean age of the females was 12 years 7 months, and of the males 12 years 5 months (see Table 3). They were all patients that had been referred to either Victoria Hospital or Edinburgh Dental Hospital and were included sequentially if they fulfilled the agreed criteria on age, having no full transverse cusp bilateral crossbite and they had not undergone surgery to the tonsils, nasal passages or adenoids. As far as possible there was matching of the proportions of males and females within each group (see Figs 5, 6).

All subjects included in the study be they anomaly or control were of the same Northern European racial background.

Ethical approval for the study was obtained (see Appendix).

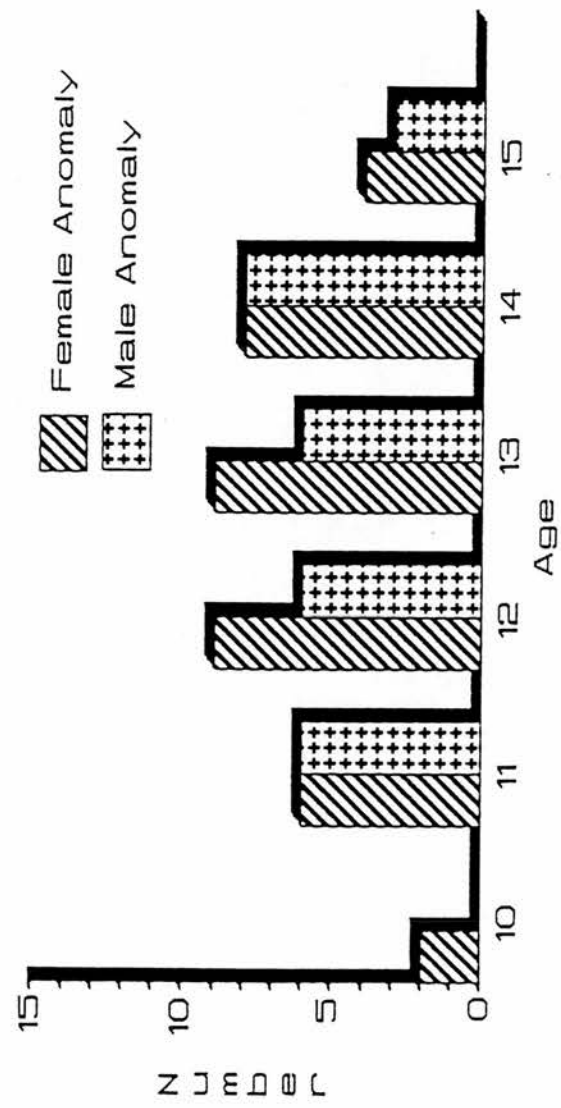
The procedures involved in the study were those normal clinical procedures that would be undertaken for any child, the only exception being the use of non-invasive rhinomanometry on the control sample. All procedures were fully explained to the patients and to their parents and approval requested before continuing. There were no refusals.

TABLE 3

Age Distribution in each Group			
	Number	Mean Age in Years	Standard Deviation
Male control	12	12.46	1.20
Male anomaly	31	12.93	1.32
Female control	24	12.71	1.39
Female anomaly	41	12.79	1.35
Male and Female control	36	12.59	0.18
Male and Female anomaly	72	12.86	0.10

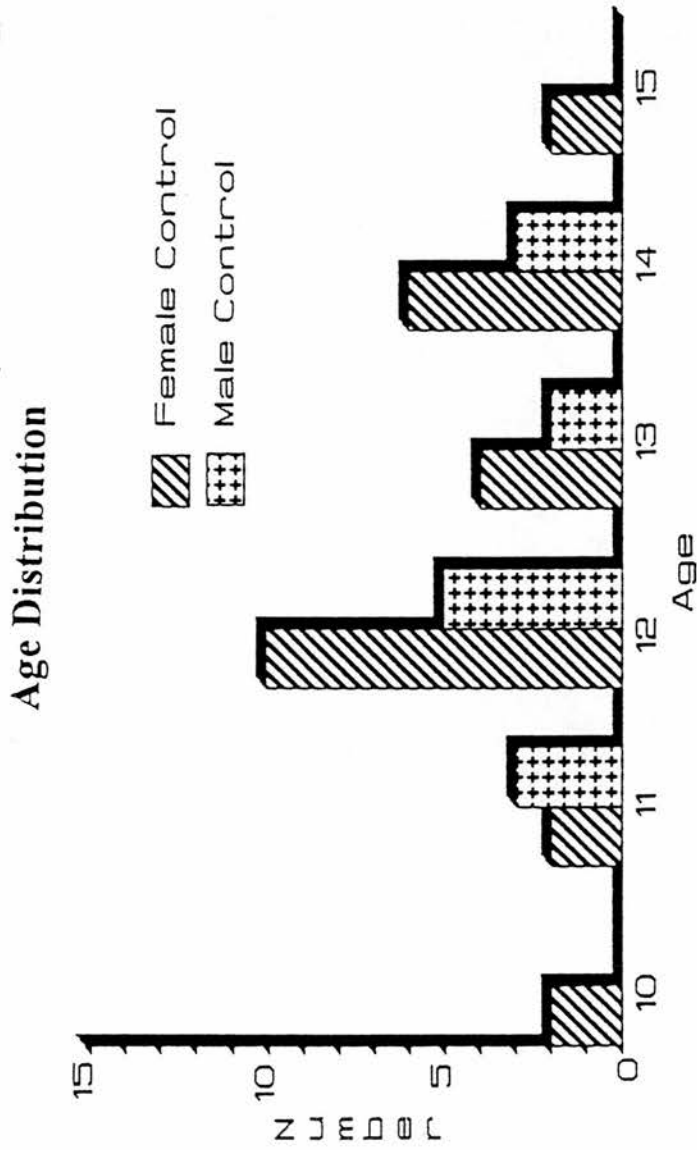
Fig. 3

Age Distribution



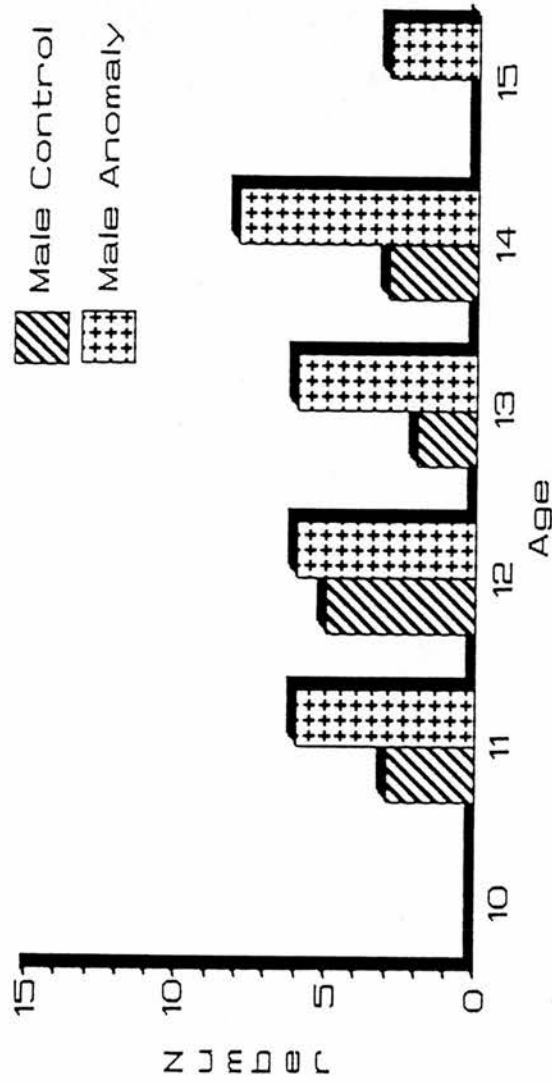
Distribution of the ages in years of 31 male and 41 female anomaly subjects.

Fig. 4



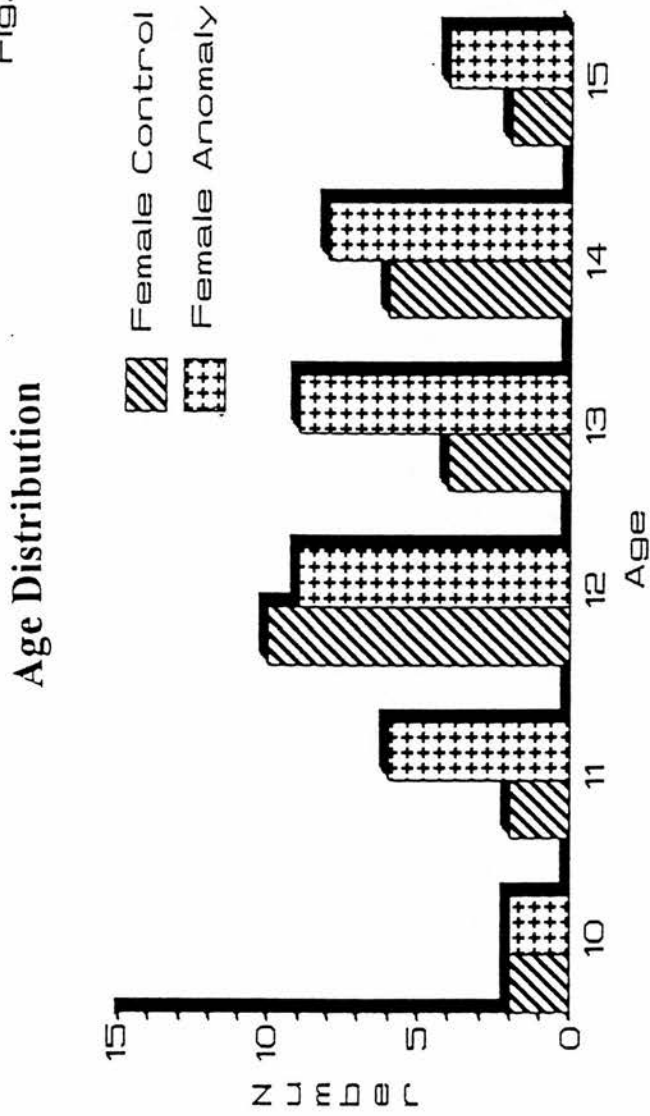
Distribution of the ages in years of 12 male and 24 female control subjects.

Age Distribution Fig. 5



Distribution of age in years of 12 male control subjects and 31 male anomaly subjects.

Fig. 6



Distribution of age in years of 24 female control subjects and 41 female anomaly subjects.

3.2 Rapid Maxillary Expansion

The anomaly sample subjects were treated as soon as the clinical necessity arose, both in terms of orthodontics and of nasal airway insufficiency. Orthodontically this normally meant that the maxillary cuspids had erupted, enabling a full transition from rapid maxillary expansion to fixed appliances in order to complete the treatment.

Full baseline records had been obtained at Tim.1 - study models, clinical photographs, PA, OPG and lateral cephalometric radiographs together with rhinomanometric measurements. The rapid maxillary expansion appliance was then constructed. This took the form of a silver/copper alloy splint giving full tooth coverage of 6543/3456, to which were soldered double buccal tubes with hooks. (See Figs 7,8). The tubes were placed anteriorly in 43/34 region to reduce the length of unsupported wire used to align the upper incisors in the later fixed appliance phase. In addition there were holes in the cast splint to facilitate its easy removal. The essential rigidity of the appliance was enhanced by palatal acrylic coverage, and the active expansion provided by a Hiram screw normally 18mm long but in small mouths 11mm.

Modifications of the appliance were used depending on the orthodontic classification of the anomaly patient. (see Fig 7).

In the twenty four Class I anomaly subjects, the standard appliance was used. (see Fig 8,9).

In the ten Class II anomaly subjects, headgear tubes were added to the buccal archwire tubes to facilitate the distalisation of the maxilla once rapid palatal expansion had been undertaken (MacNamara 1988). (see Fig 10).

In the thirty eight Class III anomaly subjects, an additional hook was placed mesial to the archwire tube to allow a protraction facemask to move the maxilla forwards relative to the cranial base (MacNamara 1988). (see Fig 11).

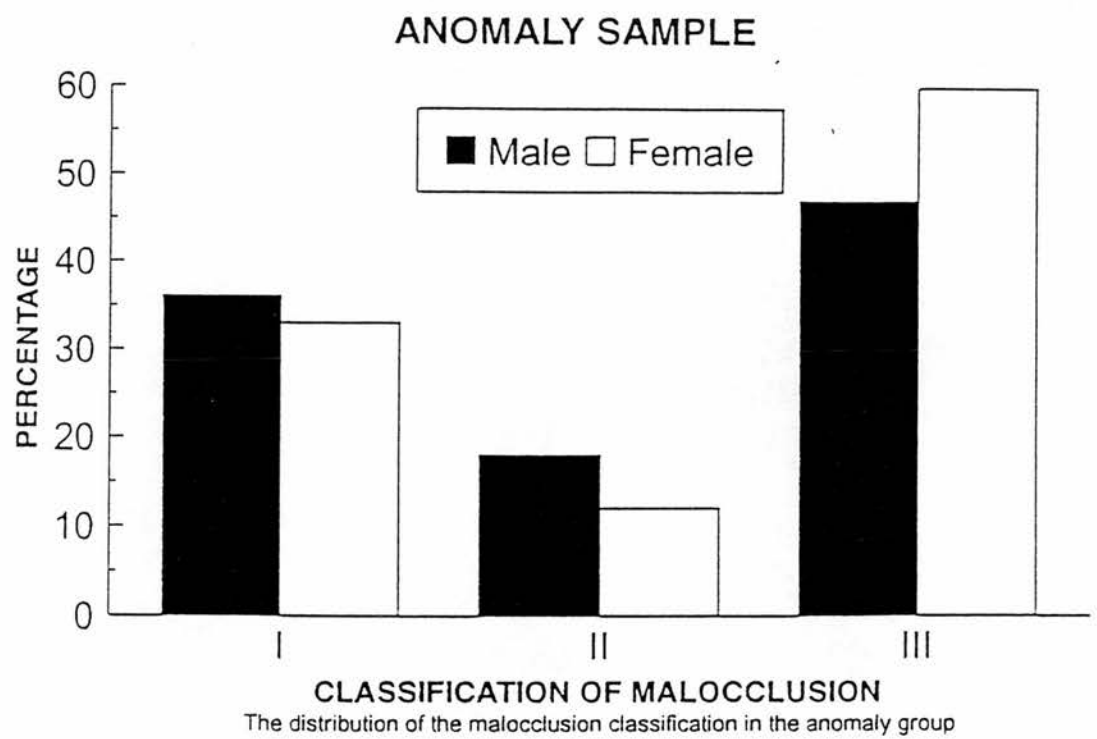


Fig. 7

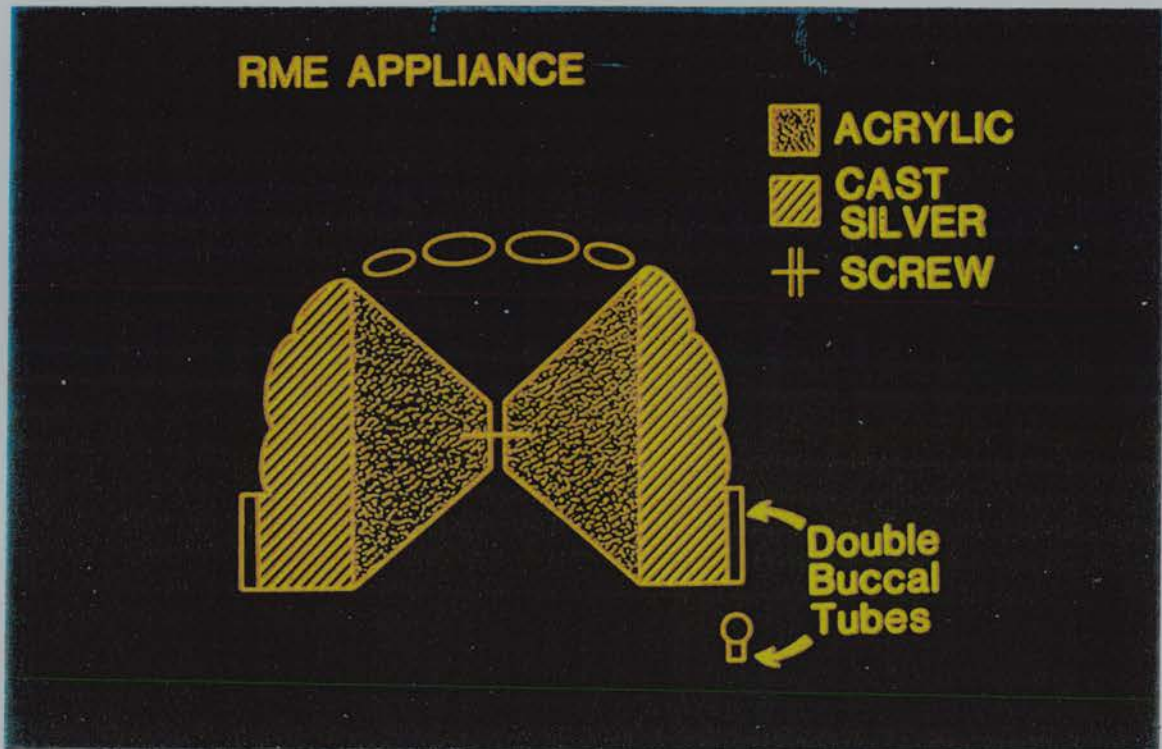


Fig. 8 RME appliance diagram

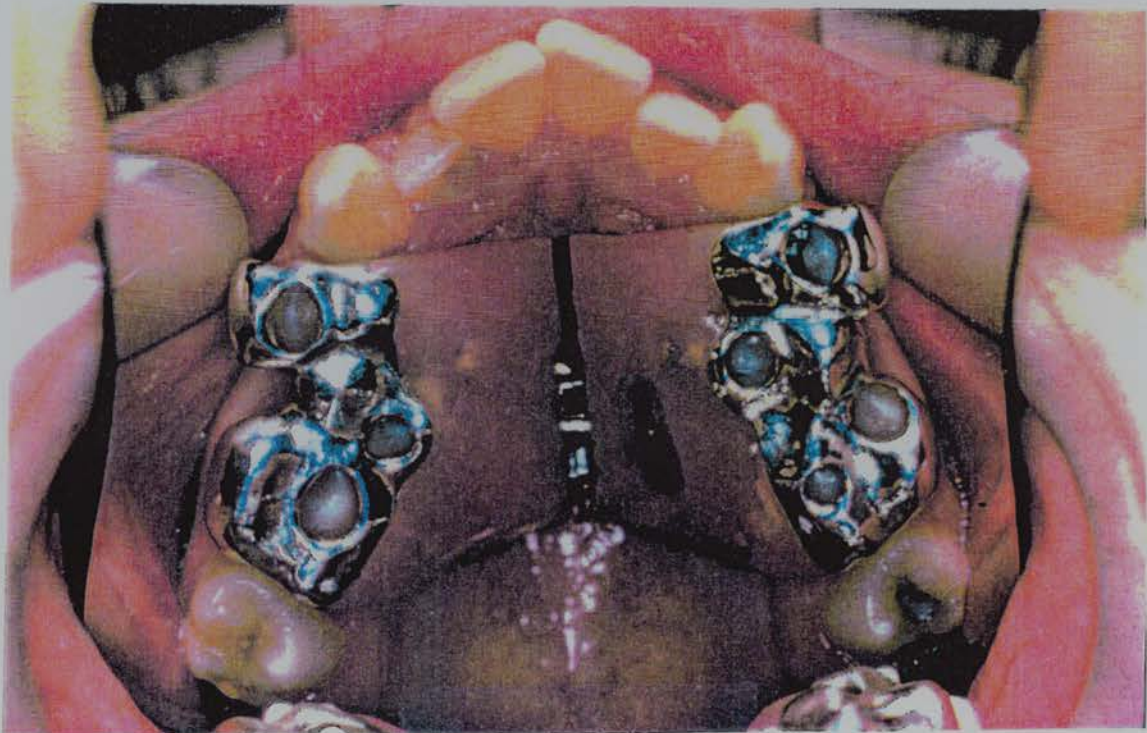


Fig. 9 RME appliance in situ

R.M.E. APPLIANCE + HEAD GEAR.

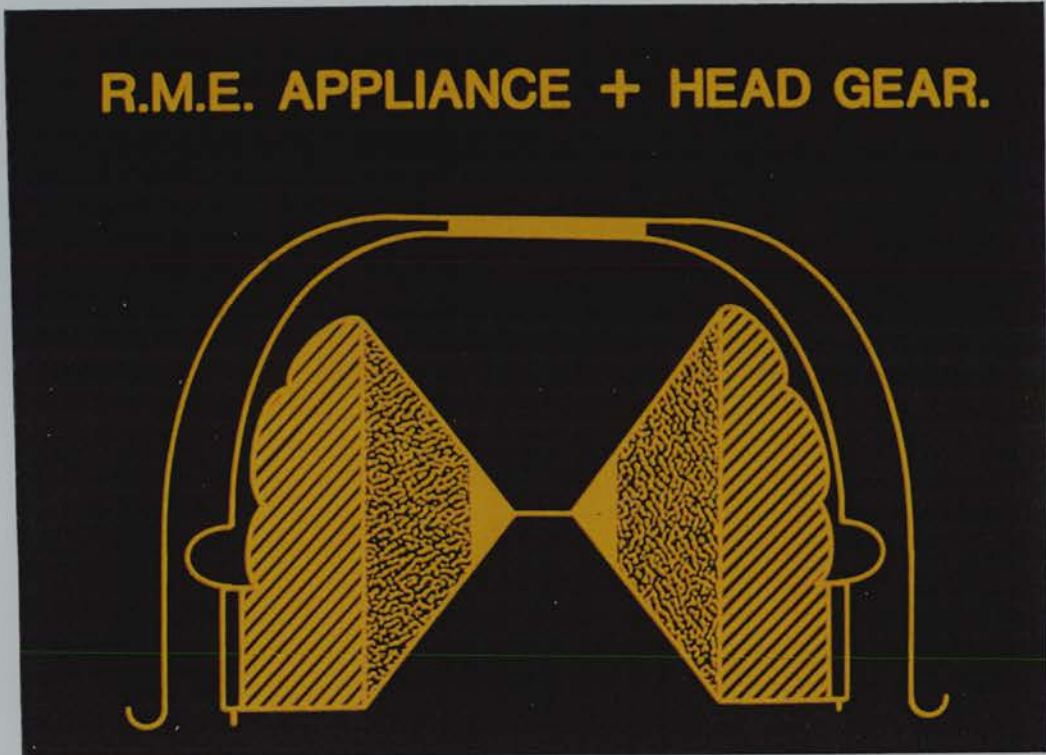


Fig. 10

RME appliance with headgear

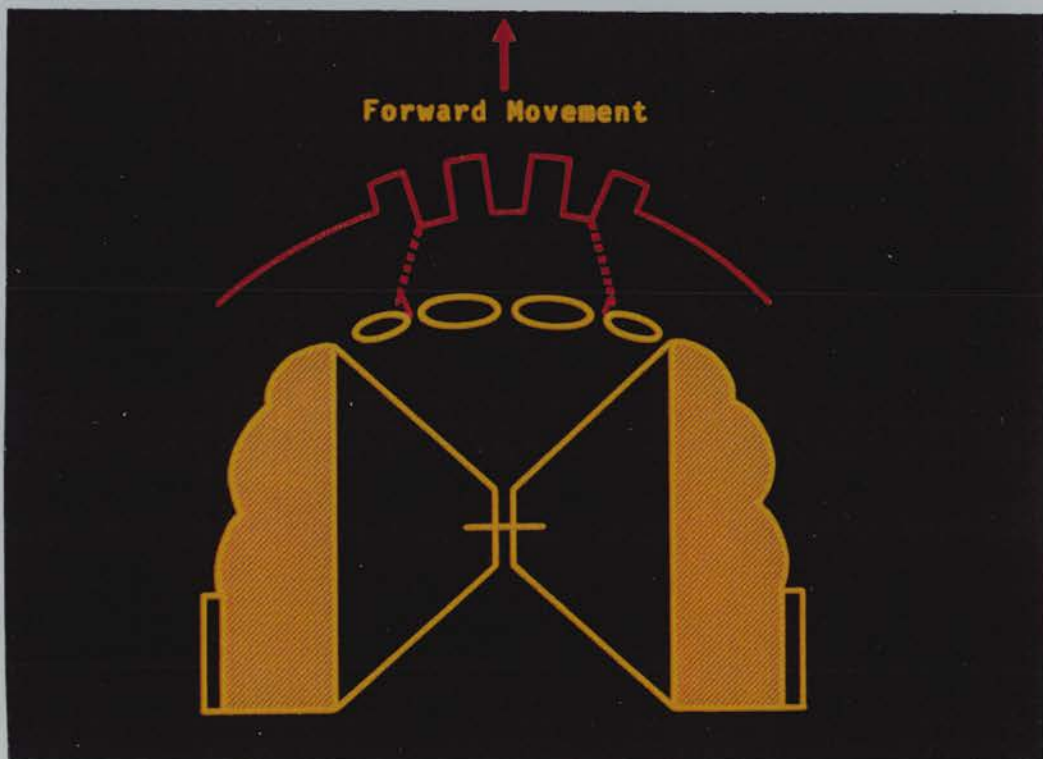


Fig. 11

RME appliance with protraction facemask

The cast splint, unlike either the Quadhelix or Biederman, is the only appliance that gives the rigidity necessary to splint the maxillary midline suture without undue flexing of the maxillary segments or undue tipping of the maxillary teeth.

Once the appliance was checked for fit, the teeth were cleaned by the Dental Hygienist and the splint cemented with glass ionomer cement. (see Fig 12). After one day, the parent (normally) activated the appliance:

- three times per day for the first week (after breakfast, on return from school and before bed)
- two times per day for the second week (morning and evening)
- one time per day for the third week, or until the bilateral crossbite had been slightly overcorrected.

The patient and parent were warned about the midline diastema that appeared between the central incisors. The active part of the rapid maxillary expansion phase of the treatment took on average 3.75 weeks, with a range from 2.25 weeks to 5.5 weeks, and a standard deviation of 0.92. At the end of this time the parents stopped turning the screw (see Fig 13), the appliance was removed, the teeth cleaned and full records obtained (Time 2). The appliance was then recemented and left non-active for three months to enable the midline suture to reossify (Ekstrom et al 1977). During this time any gross anteroposterior maxillary discrepancy was treated by taking advantage of the fluid sutures and placing headgear or a facemask as required.

The anchorage offered by the cast splint was utilised by bonding 21/12 and aligning the teeth with a series of progressively heavier archwires secured posteriorly into the tubes of the cast splint (see Fig 14). Once the incisors had been aligned, and the midpalatal suture reossified, then the cast splintage was removed, the buccal teeth cleaned and then bonded and banded. A heavy utility arch in .016" x .022" stainless steel wire was placed between 621/126 to retain the intermolar width together with

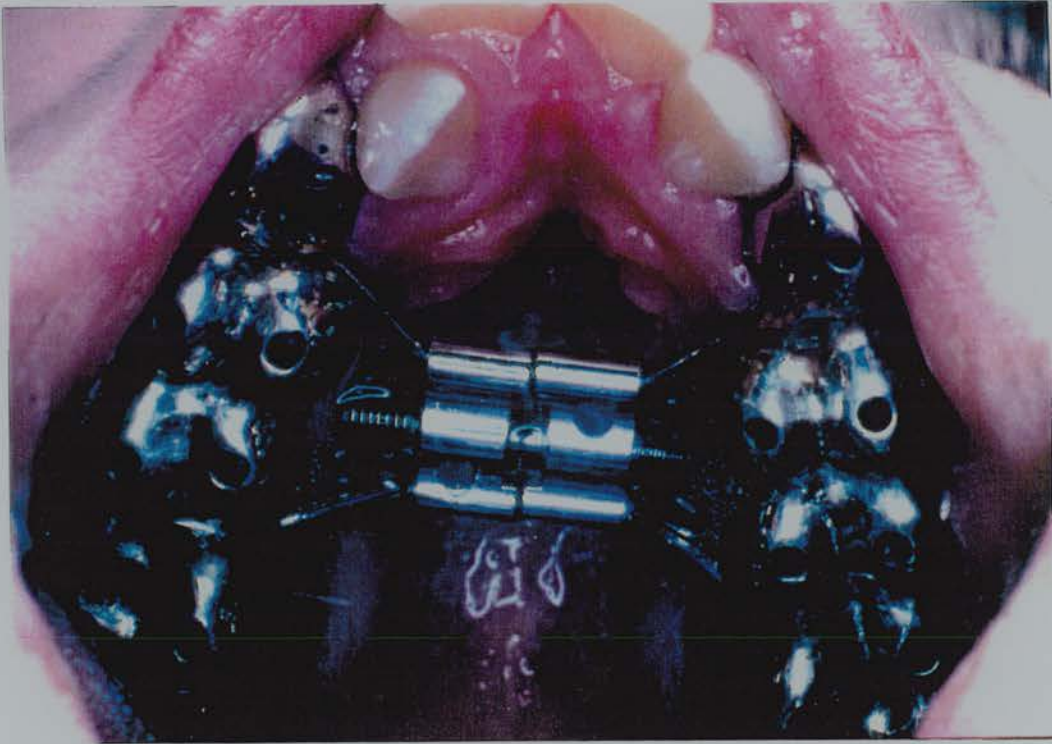


Fig. 12

RME appliance before activation

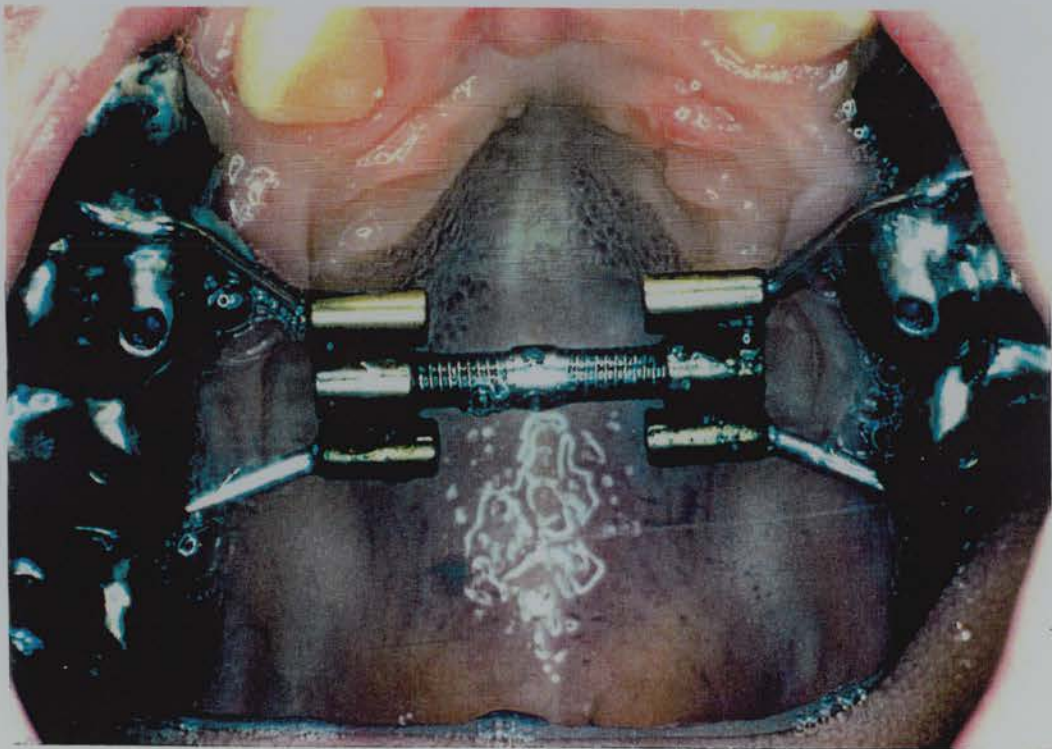


Fig. 13

RME appliance after activation



Fig. 14

RME appliance with utility (horizontal)

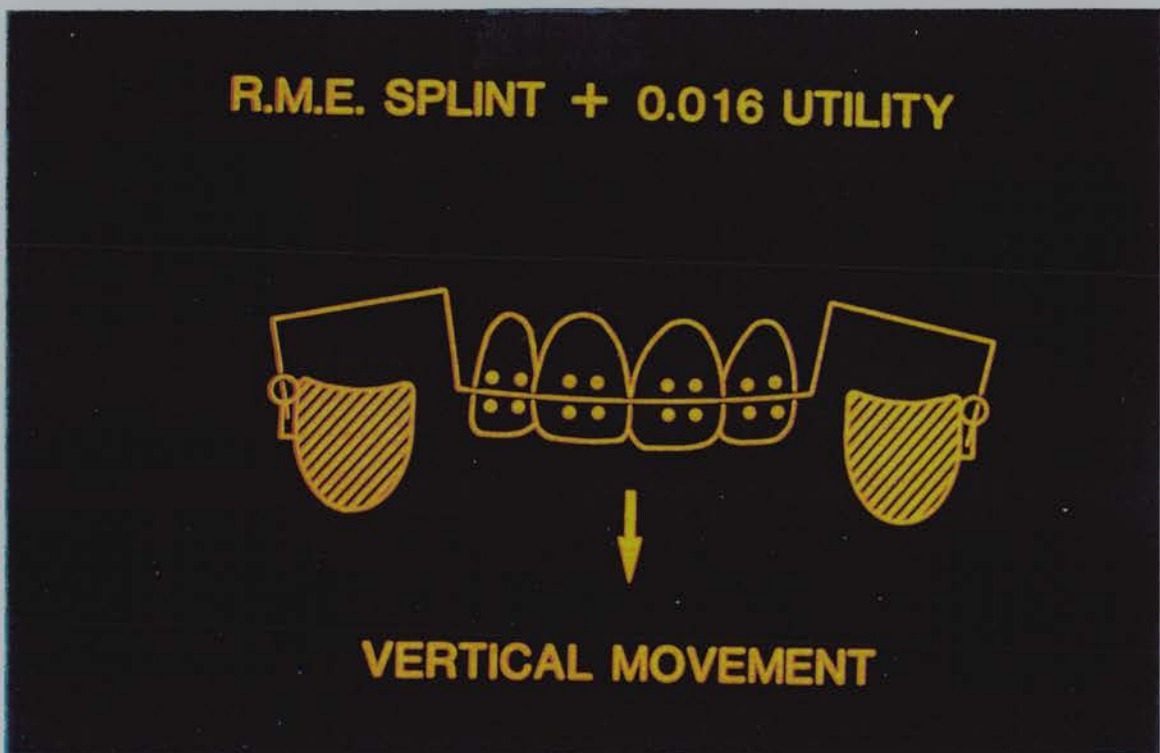


Fig. 15

RME appliance with utility (vertical)

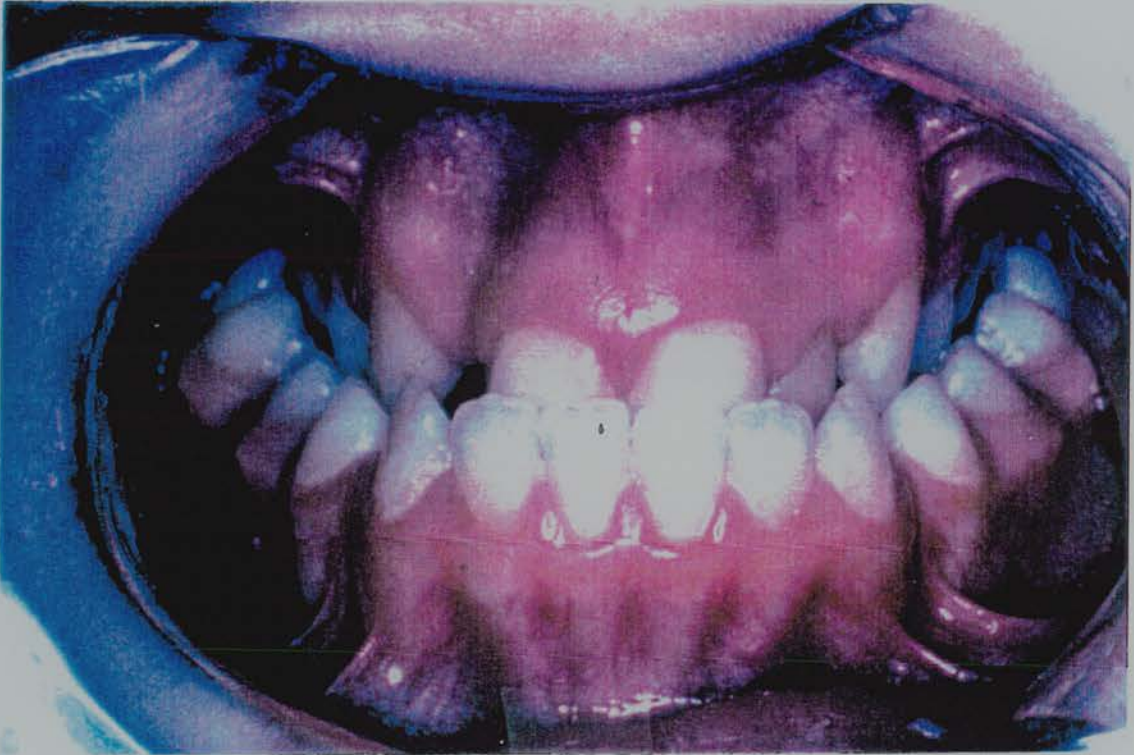


Fig. 16

Intra oral view before RME

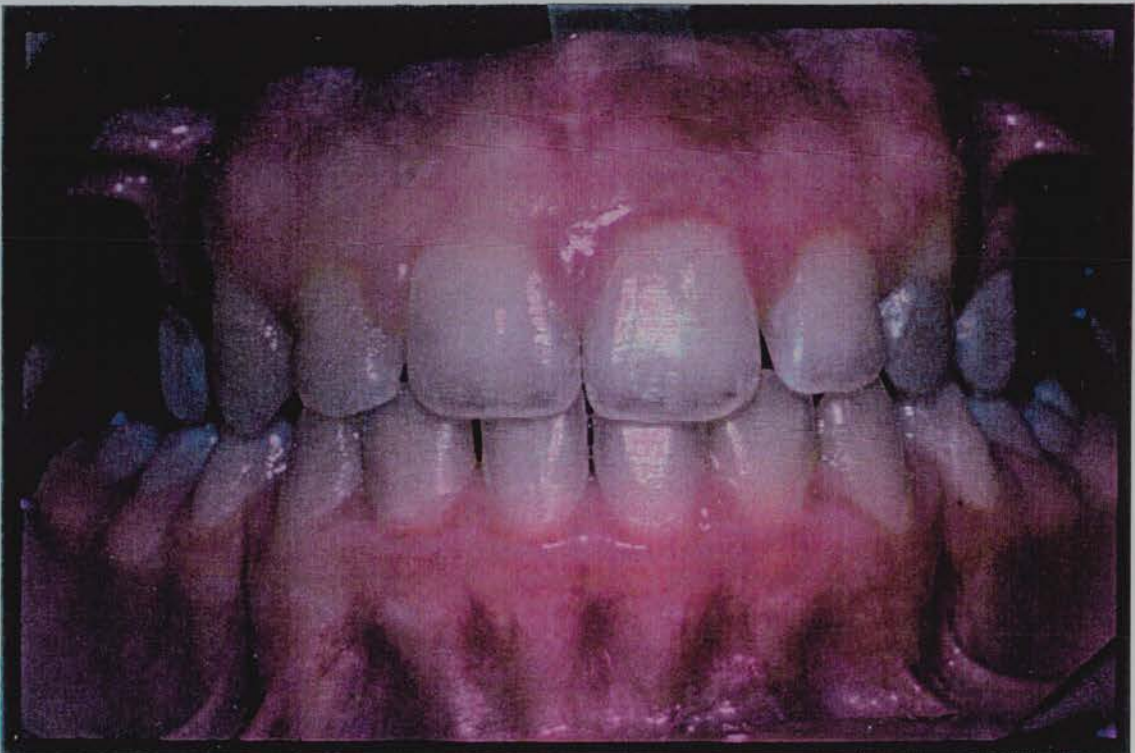


Fig. 17

Intra oral view after RME

incisal position gained (see Fig 15). This was coupled with a piggyback .012" superelastic Titanol to align the buccal segments, eventually working up through the archwires to a complete single upper arch in .018" x .025" stainless steel wire, the lower arch treatment being undertaken concurrently.

The fixed phase of treatment took in the region of one year, after which the bonds and bands were removed, the teeth cleaned and Hawley retainers fitted to both upper and lower arches to facilitate retention. These were left full-time in the region of six months, when a lower lingual fixed retainer was fitted to $\overline{321/123}$, the upper arch being retained night only by the Hawley for a further six months (see Figs 16, 17).

3.3 Measurement Procedures

Rhinomanometric Recordings

The rhinomanometer used (see Figs 18, 19) was a Mercury Electronics NR6 together with a BBC "B" Master Personal Computer printing onto an Epson FX80 Dot Matrix Printer. The software was modified for this study by the addition of a Rohrer chip to measure Laminar/Turbulent airflow (K1/K2).

The software was designed to calculate values of Nasal Airway Resistance at a pre-set pressure or pre-set flow threshold. The inspiration and expiration values were displayed on a VDU monitor for each cycle, and four such measurements were calculated, the mean values being shown on the screen. The VDU screen also displayed the pressure/flow curve from which the nasal airway resistance was calculated. This provided a visual feedback for the patient (see Fig 20).

The rhinomanometer was calibrated before each recording session utilising a dynamic pressure and flow calibration unit, which produced a flow that peaked at 150cc per second and a pressure that peaked at 500 pascal, which resembled the normal respiratory cycle and produced a sigmoid curve on the VDU. Threshold values for flow and pressure could be pre-set by adjustment to the rhinomanometer which facilitated easy and rapid calibration.

On arrival in the Department each patient whether from the control or anomaly sample, was administered Otrivine (Xylometazoline Hydrochloride) as a spray to each nostril so that any untoward effect of the nasal cycle would be eliminated. The patient was then asked to sit quietly for five minutes without blowing his or her nose.

Rhinomanometric recordings were taken thirty minutes after the nasal spray administration. The rhinomanometer had been previously calibrated before the session for a flow of 150cc per second, peaking for a pressure at 500 Pascal, in accordance with the recommendations of the International Meeting on Rhinomanometry (Clement 1984). At pressure differences exceeding this standard

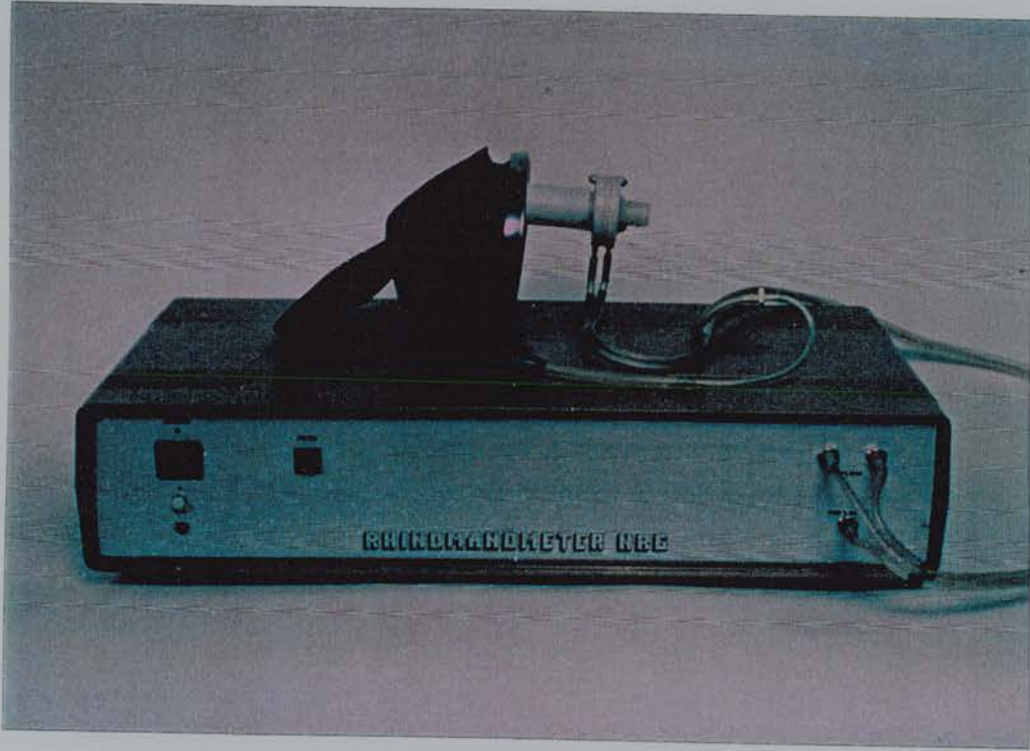


Fig. 18

Mercury NR6 Rhinomanometer

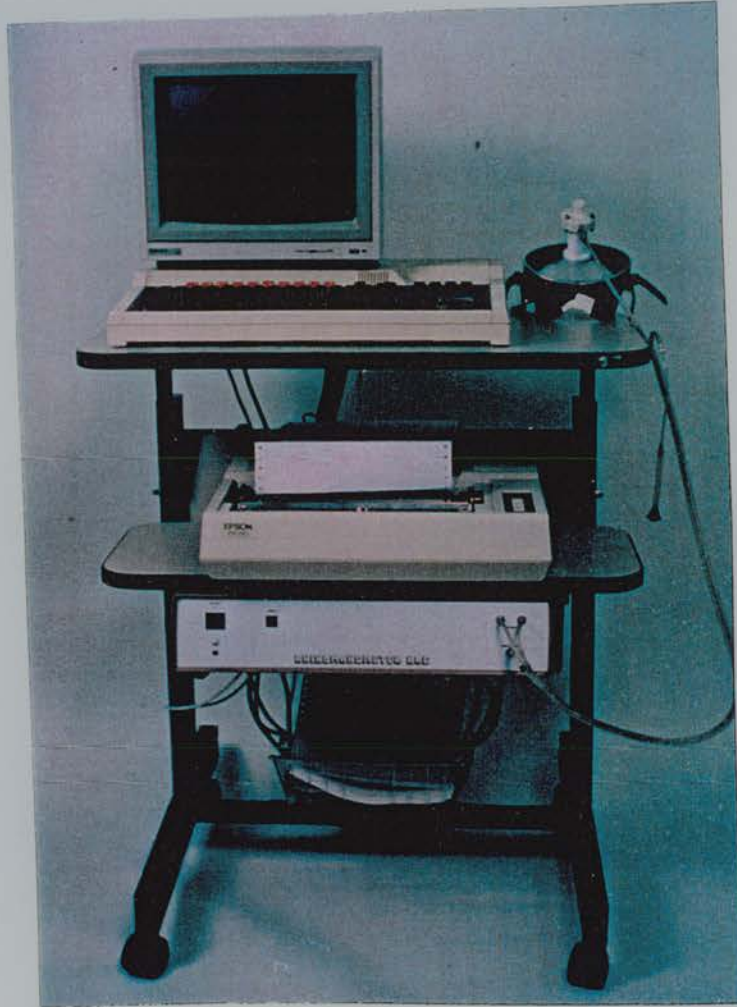


Fig. 19

NAR measurement apparatus

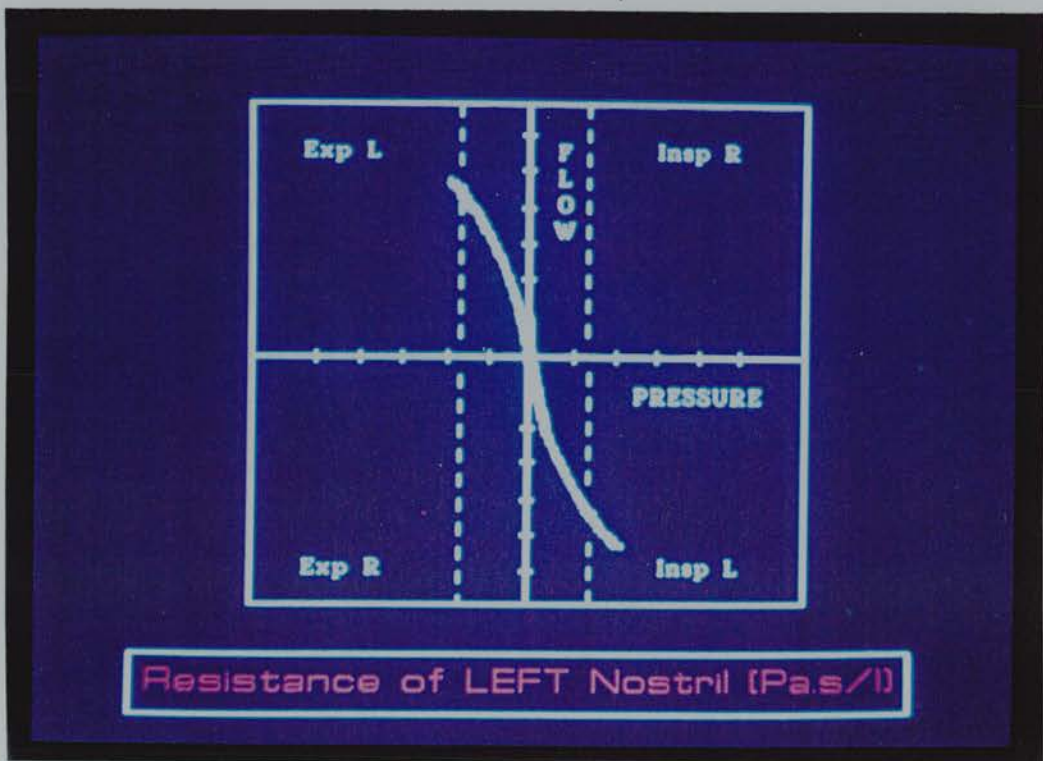
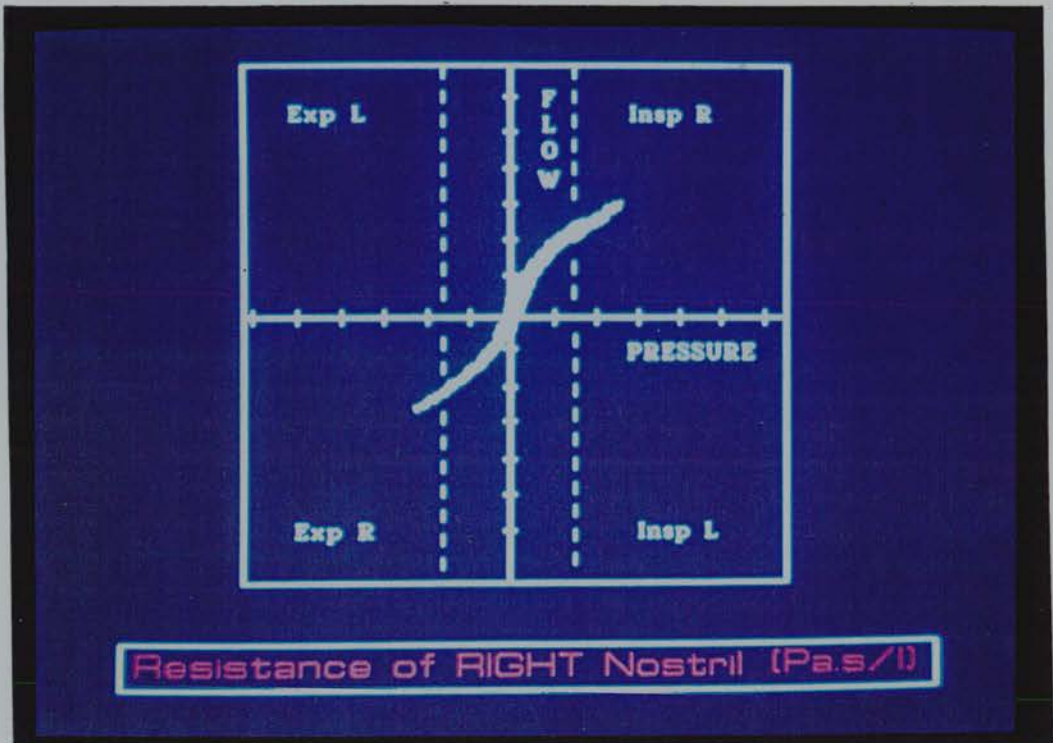


Fig. 20

Sigmoid curve on screen

threshold, the airflow and turbulence will increase and the resistance hence be higher. The laminar and turbulent component of a patient's nasal resistance are calculated separately utilising the Rohrer software chip. For each inspiration/expiration cycle a curve was fitted to all data points and the coefficients K1 (laminar) and K2 (turbulent) of the curve described by the Rohrer equation $\Delta p = K1 \times \dot{V} + K2 \times \dot{V}^2$, are calculated where Δp is the pressure drop and \dot{V} the flow. The rhinomanometer was calibrated at the start of each recording session, and the measurements of nasal resistance were made unilaterally for the right and left sides by the anterior method, as well as bilaterally by the posterior method.

Anterior method - The resistance of each nasal half was recorded as described by Broman (1982) and Solow and Greve (1980) whereby a thin tube is fixed with tape to one nostril (see Fig 21) enabling the pressure behind the opposite nasal half to be recorded against the pressure outside the nostril. In both anterior and posterior nasal resistance measurements, a scuba diving mask as recommended by Hansen et al (1984) was utilised, to cover both nose and eyes (see Fig 23). Through a hole in the screen a connector for the pneumotachograph was fitted. Soft adhesive tape was utilised to seal the nostril, a hole being punched through the tape and a flange connector pushed through from the nasal side into a short retaining tube (see Fig 22), connecting to the pressure recording tube. This arrangement enabled the pressure recording fine bore tube to be easily connected and disconnected for recording purposes (see Fig 24). The nasal airway resistance for each half of the nose was recorded by sealing the pressure tube to the opposite nostril, the pressure recorded at the nasal aperture being equal to that behind the choanae. Sixteen recordings for each component of the respiratory cycles, inspiration and expiration, were obtained. The adhesive tape was then removed from the nostril and applied to the opposite nostril, where a further sixteen recordings were gained. The mean values of the sixteen recordings were printed out to include K1 and K2. Rohrer fits the complete curve in a quadrant by least squares. The curve value for any Δp

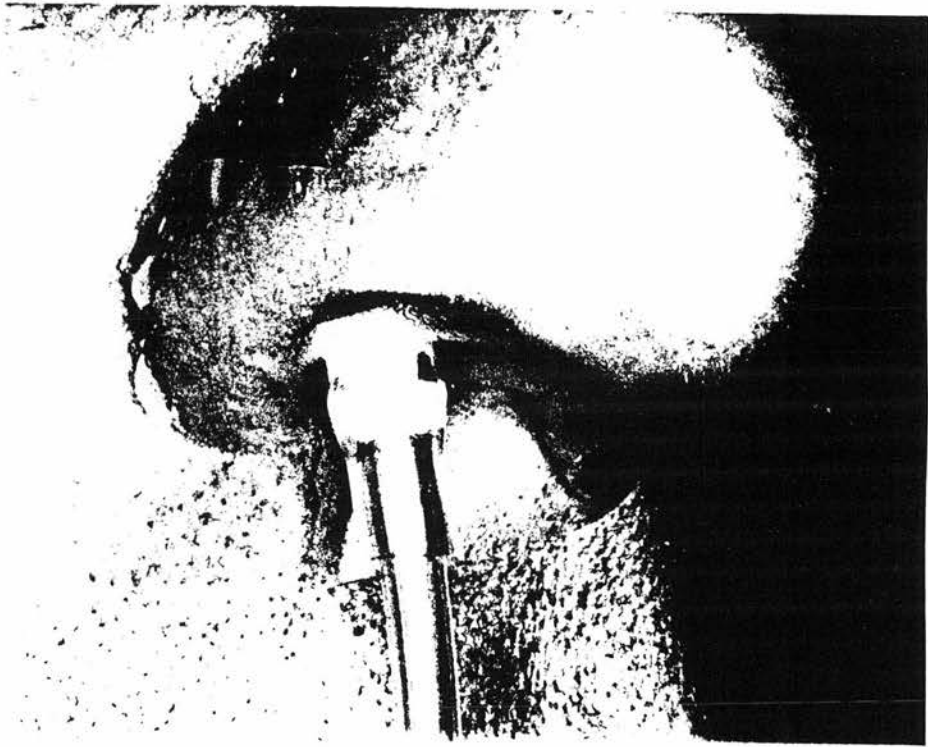


Fig. 21

Attachment of nasal tube to nostril

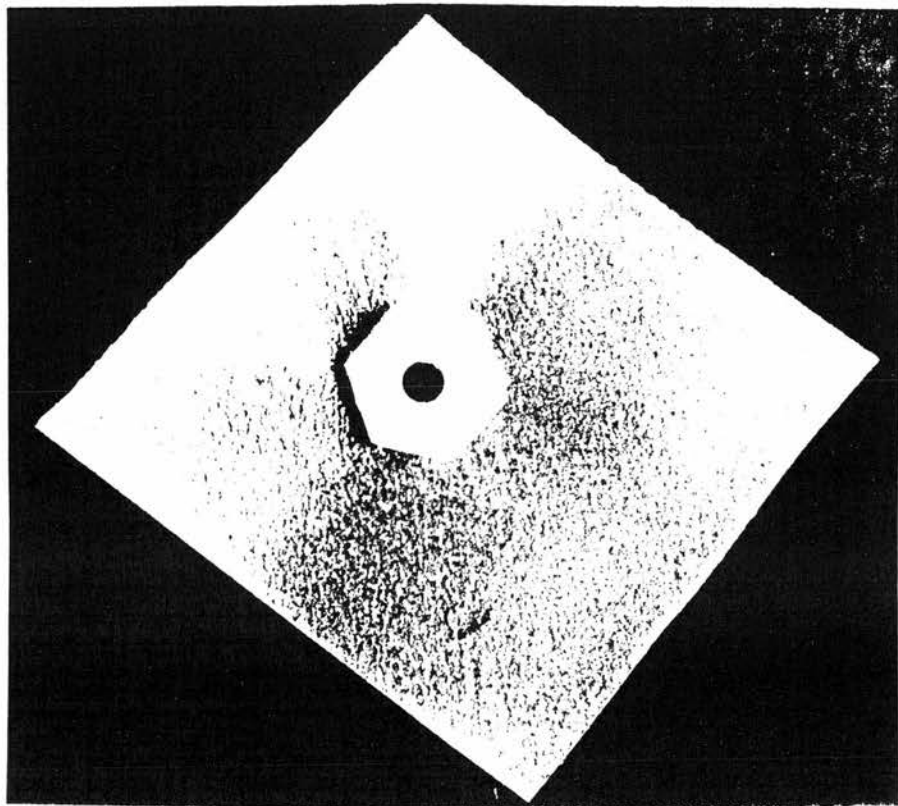


Fig. 22

Flange connector pushed through adhesive tape



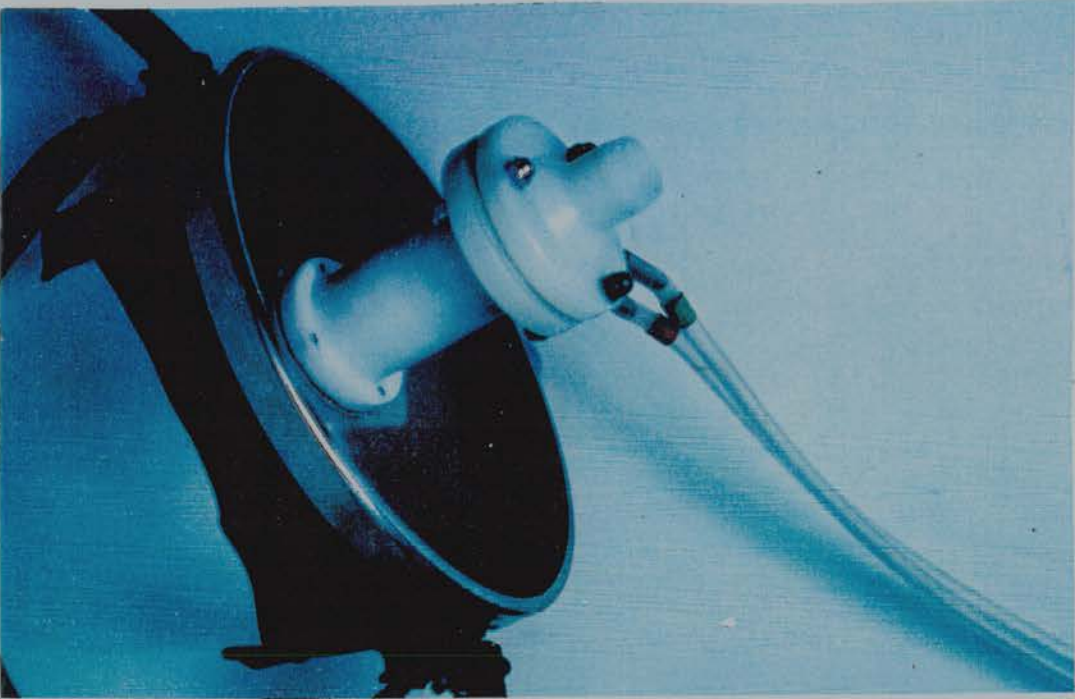


Fig. 23 Pneumotachograph and facemask

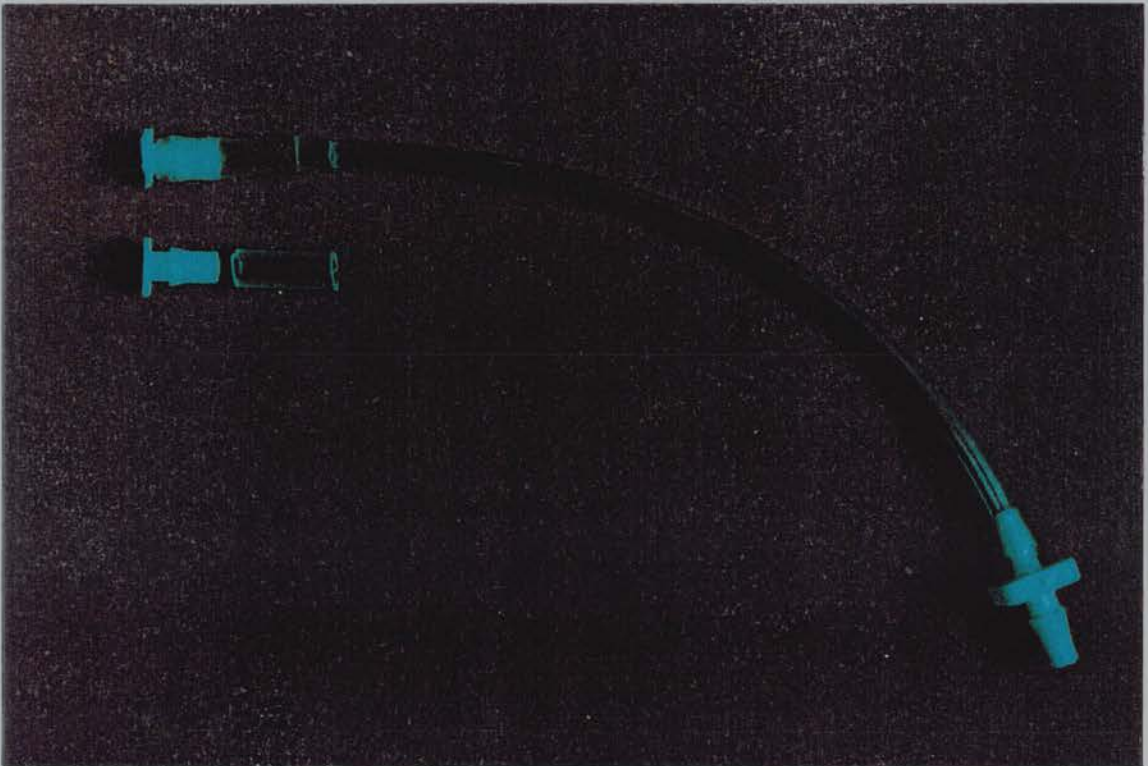


Fig. 24 Flange connector attachments



Fig. 25 Recording nasal airway resistance (posterior)

or \dot{V} (eg 150Pa) can be derived from coefficients K1 and K2. It was possible to make the recordings with little or no discomfort to the patient.

Posterior method - As reported in the literature the taking of these measurements causes rather more problems. Hence, a large diameter (10cm long x 3mm internal diameter) polythene tube modified by an otoscope tip at the end to assist with correct soft palate position, was inserted into the oropharynx to record pharyngeal pressure. Visual feedback of the monitor trace was obtained for the patient as the recording was in progress. If the trace produced on the screen became erratic this was normally due to sealing of the nose by elevation of the palate producing oral airflow and increased pressure in the tube. Some practice was required to obtain accurate posterior measurements, the visual feedback being of prime importance. Again sixteen measurements of inspiration and expiration were obtained, they being sufficient to cross the threshold line, and the values printed out to include K1 and K2. It was found that most of the subjects could perform this measurement after some training (see Fig 25).

Method error testing was undertaken on 14 subjects (6 male, 8 female), at a standardised one hour after the initial testing.

Radiographs

Standardised lateral cephalometric radiographs were taken in a cephalostat incorporated into a Morito Pan X EC orthopantomogram, modified to produce cephalometric radiographs with the long side of the film in true vertical including the whole of the subject's head. (see Fig 26).

The subject was positioned in the natural head posture utilising an eyeline mirror as described by Solow and Tallgren (1971a) with the film placed at the right side of the face. The film focus distance was 176cm, the film median plane distance was 18cm, and the enlargement was 1.1%. The exposures were made at 80kv at 0.8 seconds using Trimatic "C" T16 blue-emitting cassettes with rare earth screen and Trimax 3M blue-based fast radiographic film of 24cm x 30cm. In order to enhance image

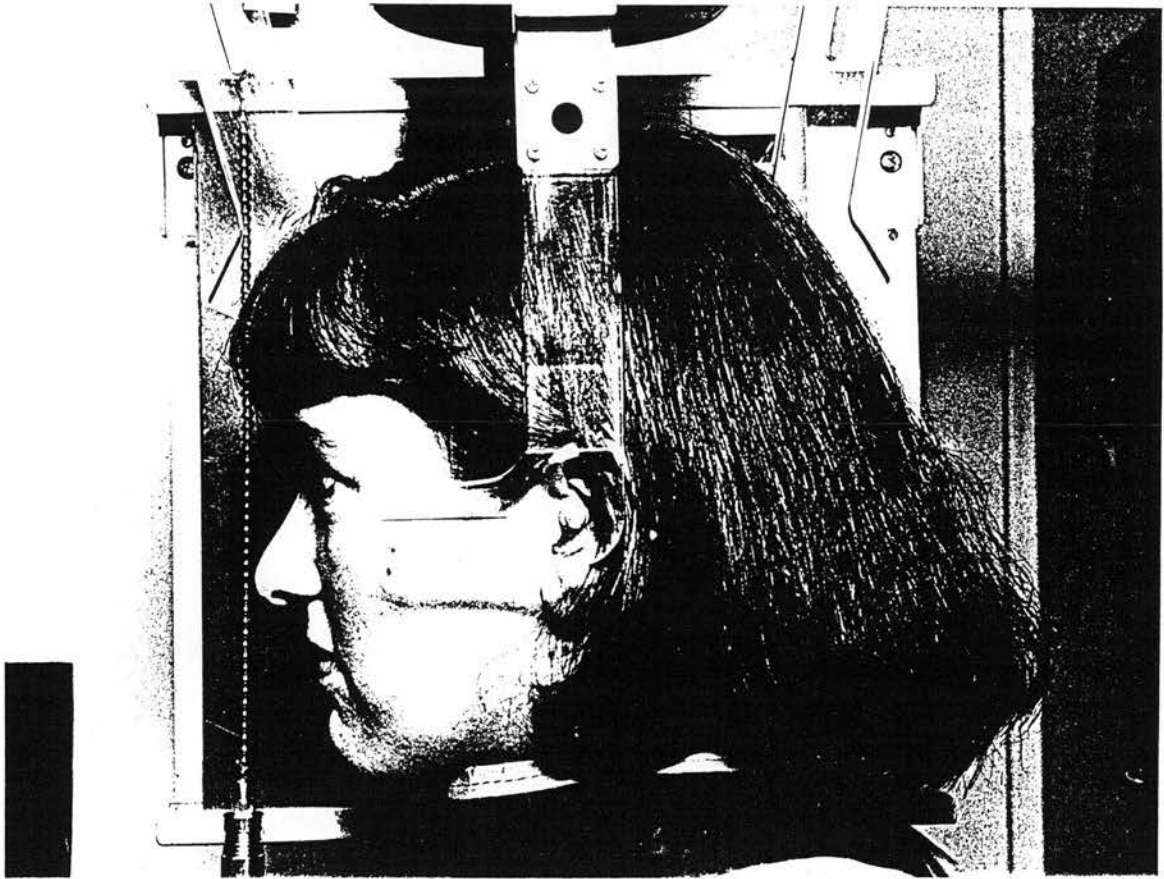


Fig. 26 Lateral skull cephalometrics in natural head position

quality, a grid was attached to the cassette. The grid had 70 absorbing strips per cm. A vertical plumb line 1.5mm in diameter was suspended at the occipital end of the filmholder in order to indicate a true vertical. A lead apron was worn by the patient to eliminate stray radiation to the body.

The posteroanterior radiographs - standardised PA radiographs were taken immediately after the lateral radiographs using the same equipment. (see Fig 27). The radiographs were taken in horizontal eye/ear line in order to avoid the projection of the par petrosa of the temporal bone. The film was exposed at 80kv for 1.3 seconds and 90mA.

The radiographs were digitised and analysed utilising software developed for the author, method error tests being undertaken as before and described in the method error section. The analysis was a 22-point analysis to show such changes in width that occur at the orbital level, the nasal cavity level, together with the maxillary and mandibular levels. In addition any changes in facial length were recorded, subdivided into nasal, maxillary and mandibular dimensions. The direction and dimensions of the movement of the nasal septum after rapid palatal expansion was noted, as were the changes in nasal dimension and maxillary dimension.

Digitiser

A GTCO digitiser coupled to a Digipad 5 screen was utilised to digitise the cephalometric and PA radiographs. Point and linearity reproducibility studies and testing of the digitiser were carried out as described in the Method Error section.

Study casts and photographs - Durable study models and working models for the RME splint were cast in stone plaster, and a registration wax bite was taken in every case. Clinical photographs were obtained at the same time to show profile, full face, three standard intraorals and a view of the palate.



Fig. 27

Postero-anterior cephalometrics

The cast splint for rapid palatal expansion was constructed in the Laboratory, fitted and activated as previously described. This took an average of 3.75 weeks. After the active part of the rapid maxillary expansion was completed, the rapid palatal expansion splint was removed, and the cephalometry, PA radiography, rhinomanometry, study models and photographs were repeated (Time 2). The splint was then refitted after the teeth had been cleaned and the patient left for some three months for the midline suture to reossify (Henrickson 1977). No method error retesting was necessary at this stage.

3.4 Measurements - Definitions and Calculations

The lateral cephalometric recordings were analysed utilising the GTCO digitiser on the Digipad 5, using software developed and written for previous studies. This gave compute-filed co-ordinate data for fifty seven reproducible points or cephalometric landmarks on the radiograph, delineating sixty four linear and angular variables. The fifty seven reproducible points were defined and numbered in the standard sequence then digitised.

- i. Reference points used in the study (see Table 4, Figs 28-30)
- ii. Radiographic cephalometric measurements (see Table 5)
- iii. Reference lines used in the study (see Table 6, Figs 31, 32)

The postero-anterior cephalometric recordings were similarly analysed using the GTCO digitiser on the Digipad 5, but here the software used was developed specifically for this study. This gave compute-filed co-ordinate data for twenty two reproducible points on the radiographs, delineating twenty ~~six~~ linear variables.

As with the lateral cephalometric recordings, the twenty two reproducible points were traced and numbered in a standard sequence before digitising.

- iv. Transverse reference point definition (see Table 7, Figs 33, 34)
- v. Transverse linear measurements (see Table 8)

3.5 STATISTICAL PROCEDURES

Statistical methods were used to analyse the distribution, the changes that occurred, and the associations and correlations found.

The formulae utilised were:

1. arithmetical mean
$$\frac{\sum x}{N}$$
2. standard deviation
$$\sqrt{\frac{\sum (x - \bar{x})^2}{N - 1}}$$
3. variance
$$\frac{\sum (x - \bar{x})^2}{N - 1}$$
4. standard error of mean
$$\sqrt{\frac{s}{N}}$$
5. variation coefficient
$$\frac{s}{\bar{x}} \times 100$$
6. variance ratio
$$\frac{s^2 \text{ max}}{s^2 \text{ min}}$$
7. paired "t" test
 - i)
$$\frac{|x_1 - \bar{x}_2|}{\sqrt{\frac{(N_1 - 1)s_1^2 + (N_2 - 1)s_2^2}{N_1 + N_2 - 2}}}$$
 - ii)
$$\frac{|\bar{x}_1 - \bar{x}_2|}{\sqrt{\frac{s_1^2}{N_1} + \frac{s_2^2}{N_2}}}$$
8. method error (Hald 1960)
$$S(i) = \sqrt{\frac{\sum (x_1 - x_2)^2}{2N}}$$
9. method error percentage
$$S(i) \% = \frac{[s(i)]}{s^2} \times 100$$
10. correlation coefficient
$$\sqrt{\frac{\sum (x - \bar{x})(y - \bar{y})}{\sum (x - \bar{x})^2 \sum (y - \bar{y})^2}}$$

TABLE 4

REFERENCE POINTS USED IN THE STUDY

ad ₃	The point on the most anterior part of the adenoidal mass the shortest distance from the posterior wall of the maxillary antrum.
ad ₂	The intersection of a line, with the adenoidal mass, drawn from pm to the mid point of a line from s-ba.
ad ₁	The intersection of a line, with the adenoidal area, drawn from pm to basion.
ai	The apex of the root of the lower central incisor.
ar	Articulare. The intersection between the external contour of the cranial base and the dorsal contour of the condylar head.
as	The apex of the root of the upper central incisor.
ba	Basion. The most postero-inferior point on the anterior margin of the foramen magnum.
cd	Condylion. The most supero-posterior point on the condylar head.
ct	Chin tangent point. The lower tangent point on the nose chin line.
cv2ap	The apex of the odontoid process of the second cervical vertebra.
cv2tg	The tangent point of OPT on the odontoid process of the second cervical vertebra.
cv2ip	The most postero-inferior point on the corpus of the second cervical vertebra.
cv4ip	The most postero-inferior point on the corpus of the fourth cervical vertebra.
ds	Dorsum nasi. The point located at the greatest convexity or concavity of the dorsum nasi.

ft	Frontal tangent point. The upper tangent point of the nose-frontal line.
gn	Gnathion. The most inferior point on the mandibular symphysis.
gn _s	Soft tissue gnathion. The soft tissue point overlying gn.
hy	Hyoideum. The most antero-superior point of the corpus of the hyoid bone.
id	Infradentale. The most antero-superior point on the lower alveolar margin.
ii	Incision inferius. The mid point of the incisal edge of the most prominent lower incisor.
int	Incision inferius. The mid point of the incisal edge of the most prominent lower central incisor.
ins	Incision occlusale. The projection of ii on OLS.
i _s	Incision superius. The mid point of the incisal edge of the most prominent upper central incisor.
Ii	Labrale inferius. The most prominent point on the prolabium of the lower lip.
Int	Lower nasal tangent point. The upper tangent point of the nose chin line.
It	Lower lip tangent point. The upper tangent point of the tangent to the lower lip through sms.
Is	Labrale superius. The most prominent point on the prolabium of the upper lip.
mlp	The posterior tangent point of ML.
mm _s	The most inferior point on the mesio-buccal cusp of the upper first permanent molar.
n	Nasion. The most anterior point of the fronto-nasal curvature.

n _s	Soft tissue nasion. The deepest point in the fronto-nasal curvature.
nst	Nasal septum tangent point. The anterior tangent point of the tangent to the nasal septum through sn.
o	Orbitale. The deepest point of the infra-orbital margin.
op	Opisthion. The most antero-inferior point on the posterior margin of foramen magnum.
p	Porion. The upper border of the bony external auditory meatus.
pg	Pogonion. The most anterior point on the mandibular symphysis.
pgn	Prognathion. The point on the mandibular symphysis farthest from cd.
pgn _s	Soft tissue prognathion. The soft tissue point overlying pgn.
pg _s	Soft tissue pogonion. The most prominent point on the chin.
pm	Pterygomaxillare. The intersection between the nasal floor and the posterior contour of the maxilla.
pr	Prosthion. The most antero-inferior point on the upper alveolar margin.
prn	Pronasale. The most prominent point on the apex of the nose.
rli	The lower tangent point of RL.
rls	The upper tangent point of RL.
s	Sella. The centre of the sella turcica.
sm	Supramentale. The most posterior point on the anterior contour of the lower alveolar process.
sme	Submentale. The deepest point in the submental neck curvature.
sm _s	Soft tissue supramentale. The deepest point in the mentolabial sulcus.
sn	Subnasale. The deepest point in the nasolabial curvature.
sp	Spinal point. The apex of the anterior nasal spine.

ss	Subspinale. The most posterior point on the anterior contour of the upper alveolar process.
ss _s	Soft tissue subspinale. The most dorsal point on the upper lip overlying ss.
sto	Stomion. The deepest point in the rima oris.
tu	Tuber. The most posterior point on the maxillary tuberosity.
tgo	The point of intersection between ML and RL.
unt	The upper nasal tangent point. The nasal tangent point of the nose frontal line.
vi	The lower point on the vertical line.
vs	The upper point on the vertical line.

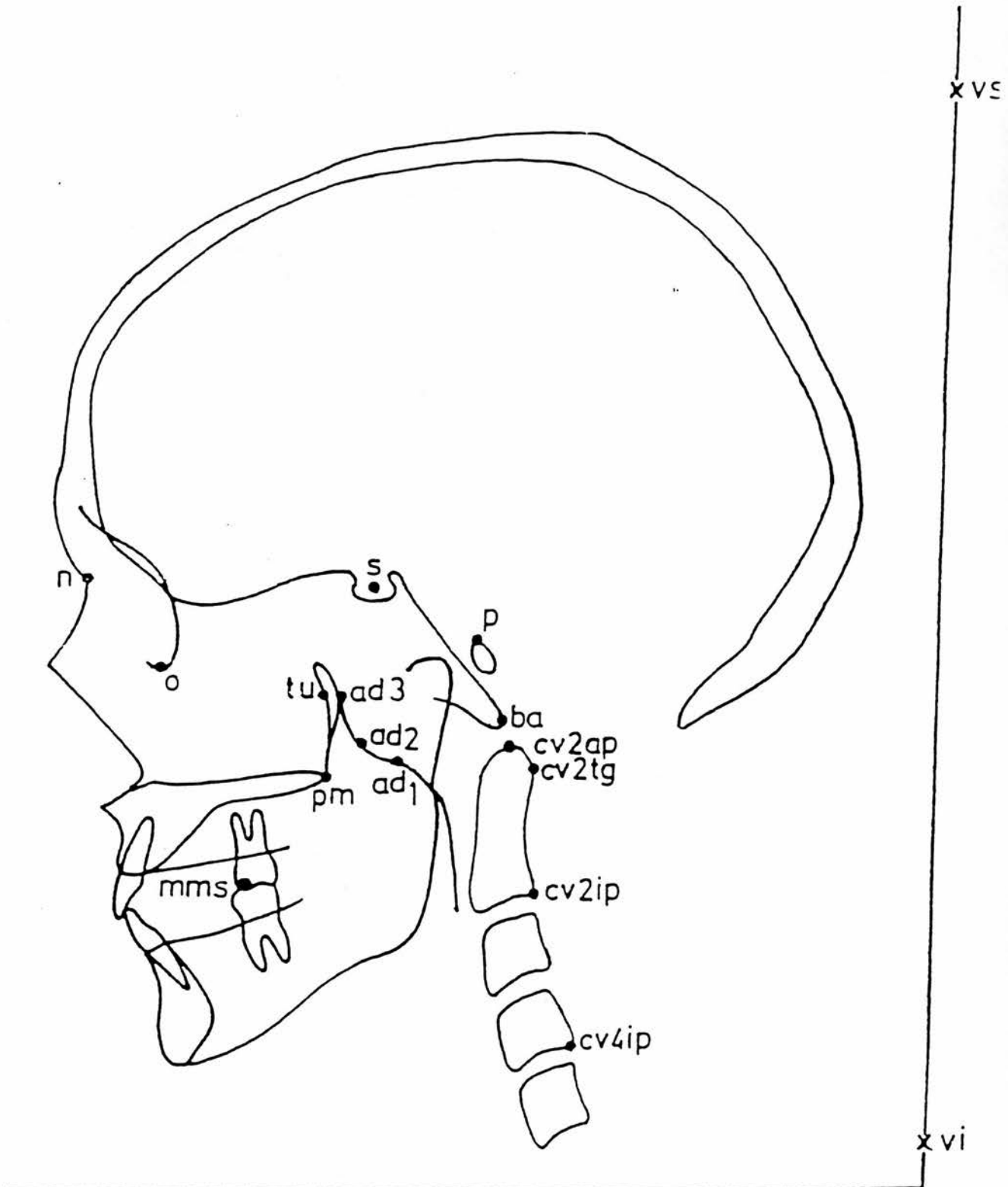


Fig. 28

Cephalometric reference points for head posture,
airway and craniofacial morphology

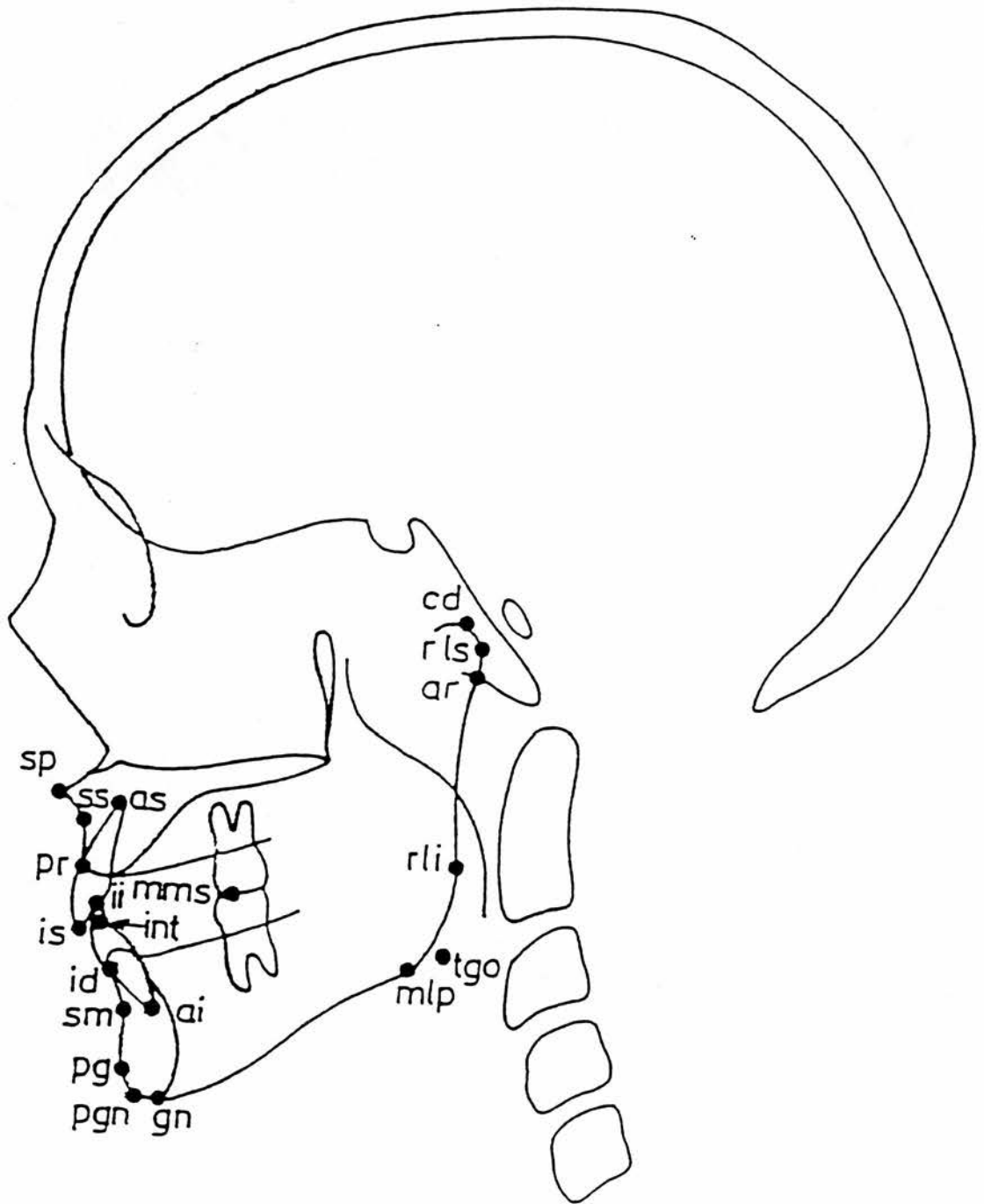


Fig. 29 Cephalometric reference points for mandibular and dento-alveolar structures

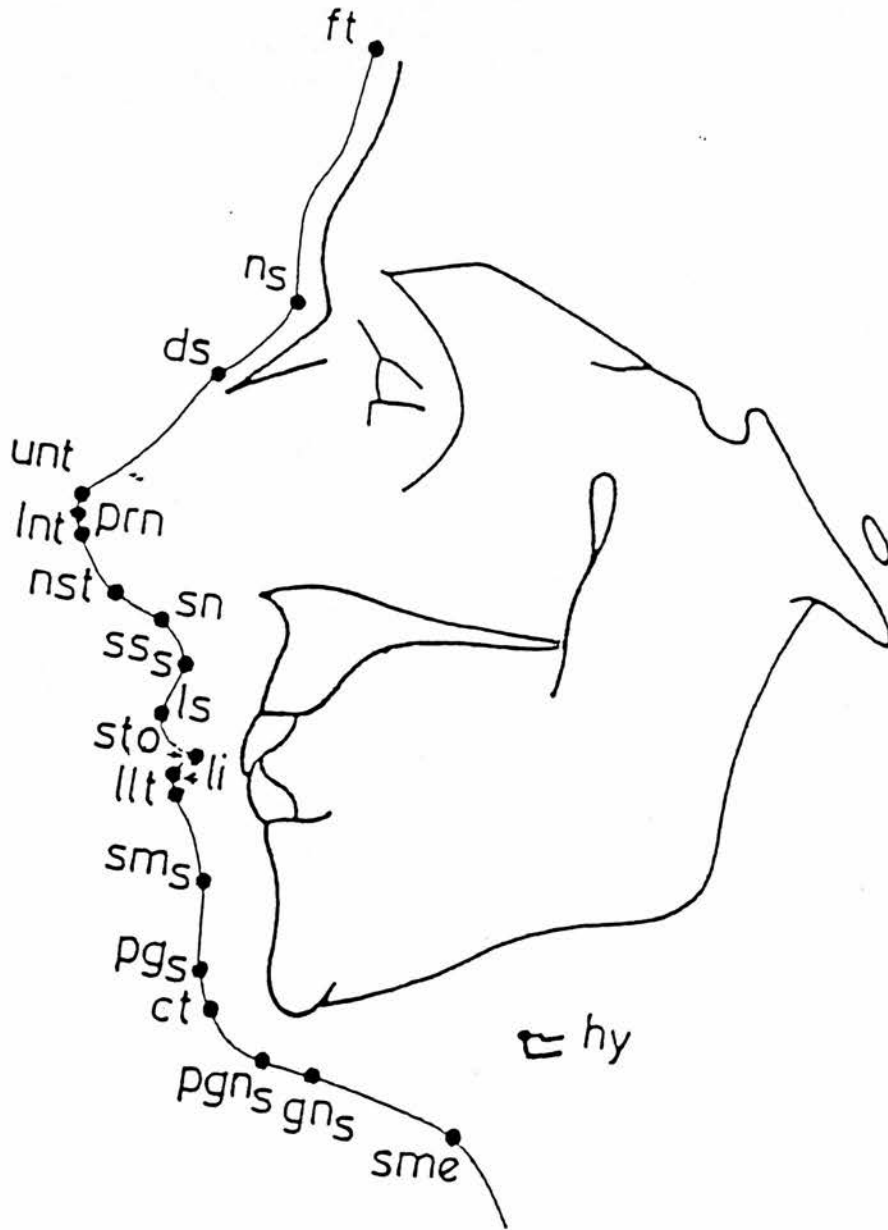


Fig. 30

Cephalometric reference points for soft tissue measurement

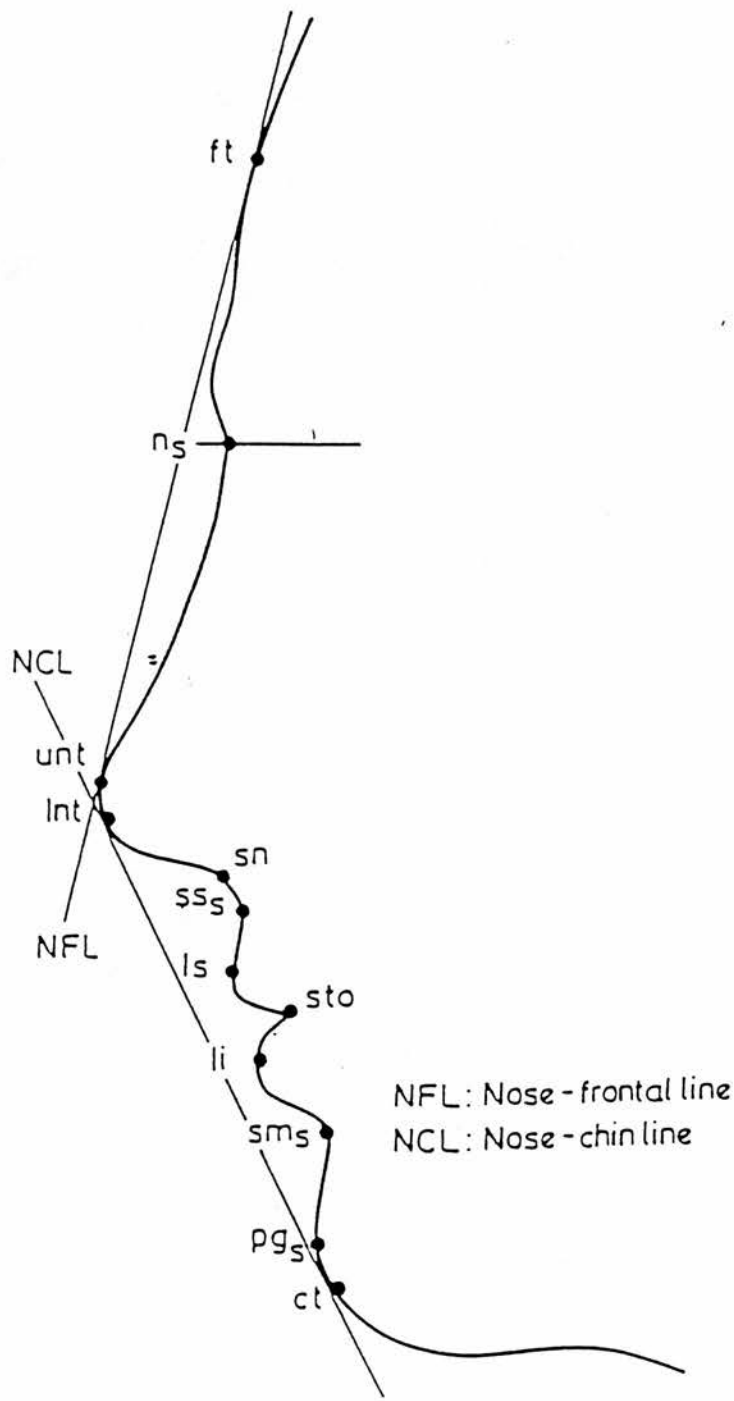


Fig. 31

Soft tissue reference planes

TABLE 5

RADIOGRAPHIC CEPHALOMETRIC MEASUREMENTS**a. Linear dimensions**

1. n-s
2. n-sp
3. n-gn
4. s-ba
5. s-ar
6. s-pm
7. s-tgo
8. sp-gn
9. ar-tgo
10. sp-pm
11. ss-pm
12. pgn-cd
13. pg-tgo
14. sp-is
15. ii-gn

b. Angular dimensions

16. n-s-ba
17. n-s-ar
18. pm-s-ba
19. s-n-sp
20. s-n-ss
21. s-n-sm

- 22. s-n-pg
- 23. ss-n-sm
- 24. ss-n-pg
- 25. NSL/NL
- 26. NSL/ML
- 27. NL/ML
- 28. NSL/MBL
- 29. ML/RL

c. Dento-alveolar relations:

- 30. IL_s /NL
- 31. IL_i /ML
- 32. oj/(mm)
- 33. ob/(mm)

d. Head posture:

- 34. NSL/VER
- 35. NL/VER
- 36. NSL/OPT
- 37. NSL/CVT
- 38. NL/OPT
- 39. NL/CVT
- 40. OPT/HOR
- 41. CVT/HOR
- 42. FH/VER
- 43. FH/OPT

44. FH/CVT

e. Airway:

45. pm-ad₁

46. pm-ad₂

47. pm-ad₃

48. tu-ad₃

f. Soft tissue:

49. n_s-sn

50. n_s-prn

51. Int to n-ss

52. s-n_s-unt

53. sto to NL

54. s-n_s-ss

55. sn to Int-I_s

56. I_s to NCL

57. sto to ML

58. s-n_s-sm_s

59. sm_s to li-pg_s

60. li-NCL

61. ss_s-n_s-sm_s

62. sto to OL_s

63. s-n_s-pg_s

64. NFL/NCL

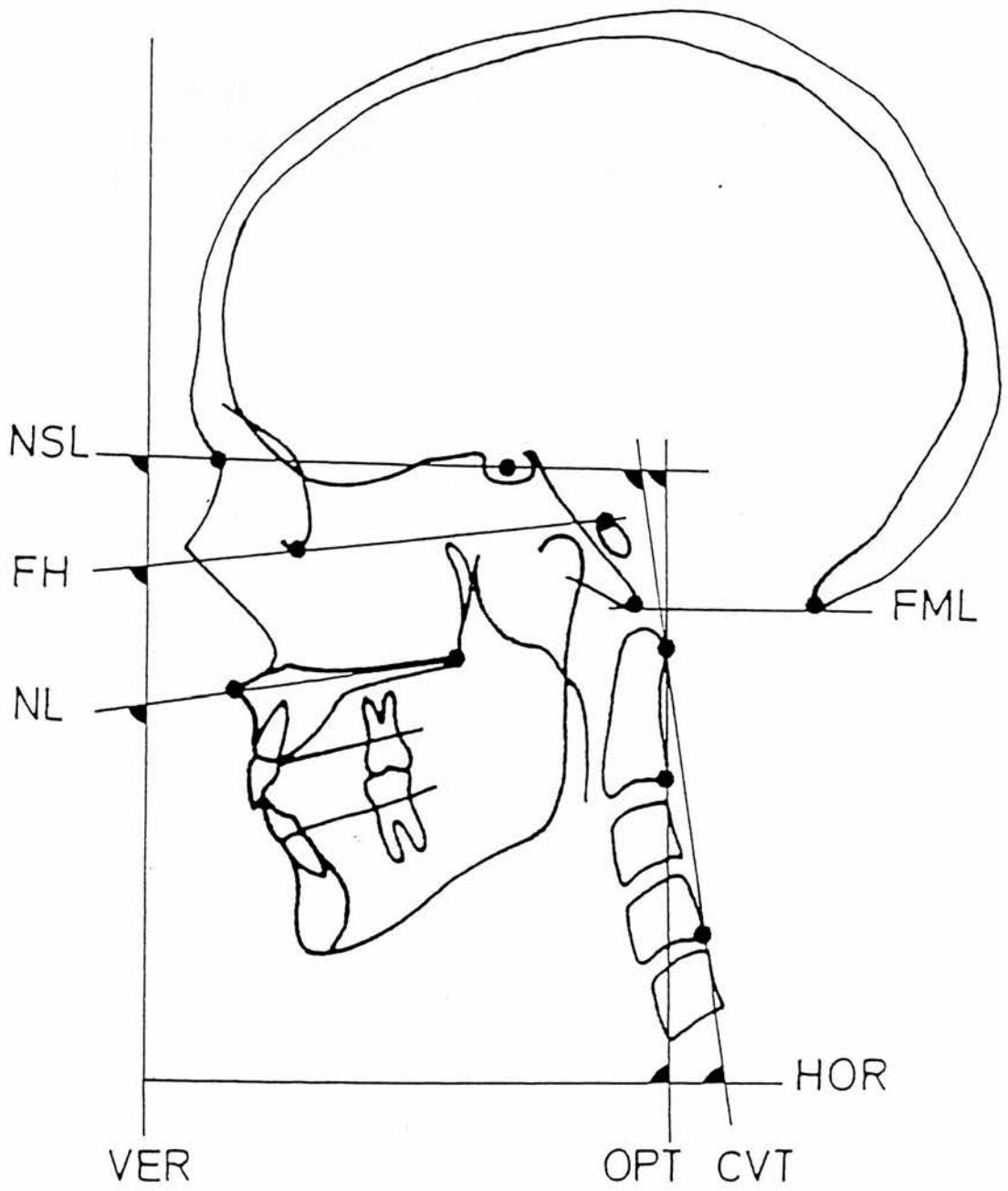


Fig. 32 Angular head posture variables

TABLE 6

REFERENCE LINES USED IN THE STUDY

CVT	Cervical vertebrae tangent. The posterior tangent to the odontoid process through cv4ip.
FH	Frankfort horizontal line. The line drawn from orbitale to porion.
HOR	True horizontal line. The line perpendicular to VER.
ob	Overbite. A linear measurement of the distance the upper central incisor overlaps the most prominent lower incisor measured from ii to int.
oj	Overjet. A linear dimension of the protrusion of the upper central incisor measured from is to int.
NCL	Soft tissue nose chin line. The line through Int and ct.
NFL	Soft tissue nose frontal line. The line through ft and un.
MBL	Mandibular base line. The line through pgn and cd.
ML	Mandibular line. The tangent to the lower border of the mandible through gn.
NL	Nasal line. The line through sp and pm.
NSL	Nasion-sella line. The line through n and s.
OPT	Odontoid process tangent. The posterior tangent to the odontoid process through cv2ip.
RL	Ramus line. The tangent to the posterior border of the mandible.
VER	True vertical line projected on the film.

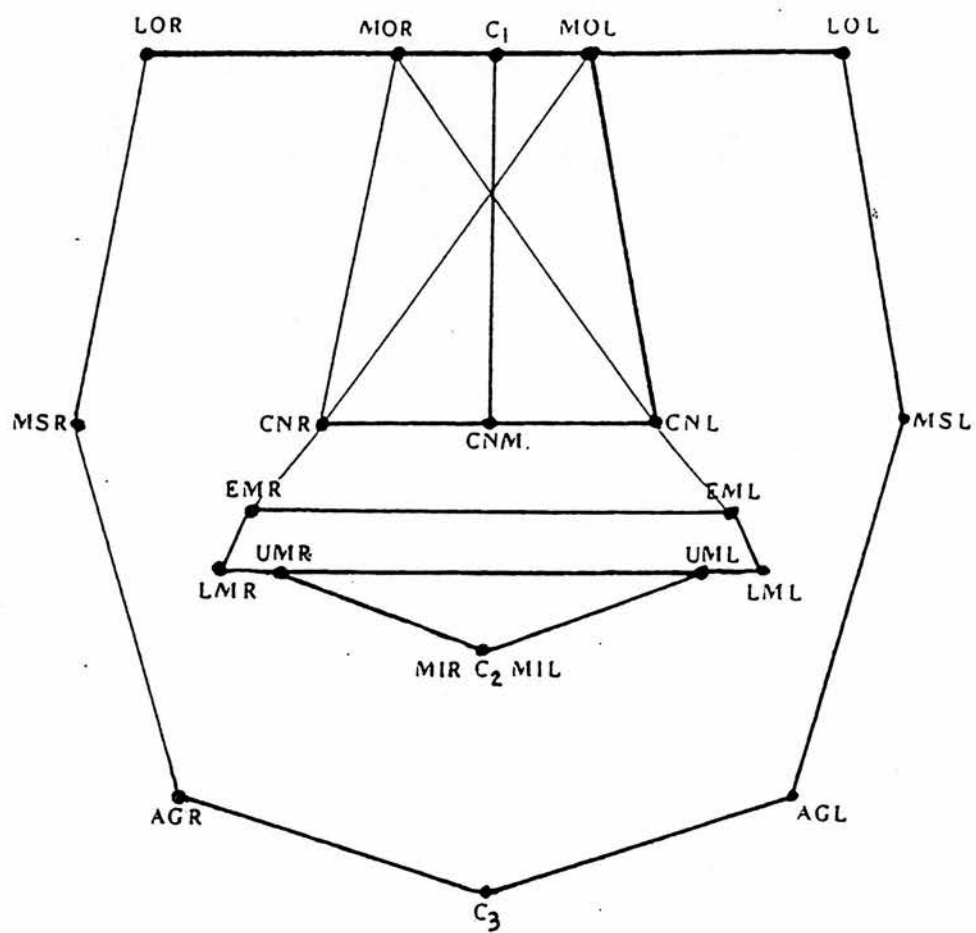


Fig. 33 Transverse cephalometric points

TABLE 7

TRANSVERSE POINT DEFINITIONDefinitions of the points used -

LOR	The most lateral point on the right orbit
MOR	The most mesial point on the right orbit
C ₁	The central point between MOR and MOL
MOL	The most mesial point on the left orbit
LOL	The most lateral point on the left orbit
MSR	The most lateral point on the right mastoid bone
CNR	The right cavum nasi
CNM	The point of the end of the nasal septum
CNL	The left cavum nasi
MSL	The most lateral point on the left mastoid bone
EMR	Ecto-maxillare right
EML	Ecto-maxillare left
LMR	The most lateral point on the lower right first molar
UMR	The most lateral point on the upper right first molar
MIR	The most mesial point on the upper right incisor
C ₂	Central point between the two central incisors
MIL	The most mesial point on the upper left central incisor
UML	The most lateral point on the upper left first molar
LML	The most lateral point on the lower left first molar
AGR	The deepest point on the right antegonion notch
C ₃	The centre point on the most inferior border of the mandible
AGL	The most lateral point on the left antegonion notch

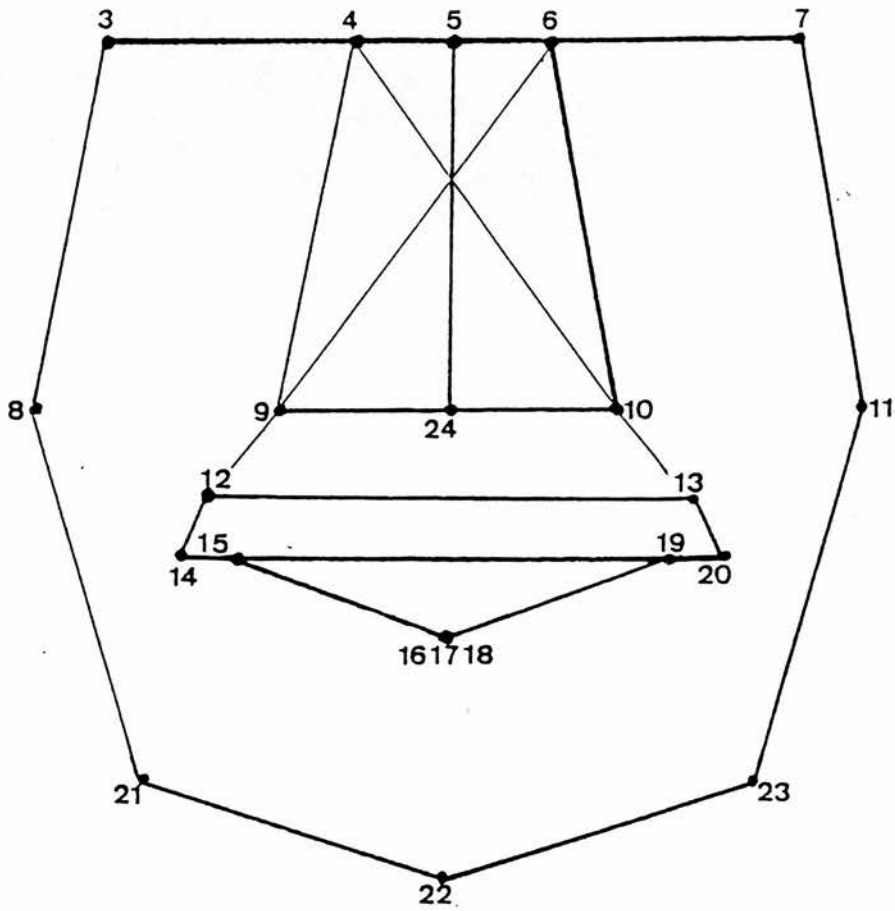


Fig. 34 Transverse cephalometric points in numerical sequence

TRANSVERSE LINEAR MEASUREMENTSat orbit level:

1. LOR-LOL
2. MOR-MOL
3. LOR-UMR
4. LOL-UML

at nasal level:

5. MSR-MSL
CNR-CNL
6. CNR-CNM
CNM-CNL
7. MOR-CNR
8. MOL-CNL

at maxillary level:

9. EMR-EML
10. UMR-UML
11. MIR-C₂
12. C₂-MIL
13. MIR-MIL
14. CNR-EMR
15. CNL-EML
16. EMR-UMR

17. EML-UML

18. UMR-MIR

19. MIL-UML

at mandibular level:

20. LMR-LML

21. AGR-AGL

proportionality:

22. NASDIM - 4-10, 6-9

23. MAXDIM - 4-13, 6-12

24. MANDDIM - 4-23, 6-21

The linear and angular measurements from both the cephalometric and PA measurements were analysed using an IBM 286 Computer and the in-house calculation package which had been rewritten for an IBM. The data was then downloaded onto the SPSS-X (Statistical Package for Social Studies) mainframe of Edinburgh University for final analysis.

In the statistical analysis, a probability $p < 0.05$ or less was interpreted as statistically significant, $p < 0.01$ rather more so and $p < 0.001$ highly significant.

4.

METHOD ERRORS

4.1 RHINOMANOMETRIC METHOD ERRORS

To ascertain the rhinomanometric method error, fourteen subjects (6 male, 8 female) from the anomaly sample had duplicate airway assessments measured at the same recording session, with an interval of at least one hour between the initial and the duplicate measurements, under the same experimental conditions.

Measurements were made for right and left unilateral nasal resistance using the values obtained by the anterior method, as well as total nasal resistance using recordings obtained by the posterior method.

Results

The rhinomanometric method errors are given in Table 9.

There were no systematic differences shown at the $p < 0.05$ level. The method errors ranged between 14.79 for the posterior expiration and 37.91 for the anterior inspiration (left). These values compared well with results of previous studies (see Table 10).

The error percentage ranged between 3.57% for the anterior expiration (right) and 11.76% for the anterior inspiration (right).

TABLE 9

METHOD ERRORS - NASAL AIRWAY RESISTANCE
 DUPLICATE DETERMINATION OF NRR FOR POSTERIOR AND ANTERIOR MEASUREMENTS
 (Pascal/cc/sec x 10⁴)

Variable	N	Min.	Max.	Mean Diff.	Standard error of Mean Difference	P	S(i)	S(i)%
ANT(L)								
INSP	14	-202	256	14.10	11.39	ns	37.91	10.13
EXP	14	-78	125	18.02	11.70	ns	38.48	9.50
ANT (R)								
INSP	14	-146	82	19.80	10.92	ns	35.90	11.76
EXP	14	-45	49	2.40	4.55	ns	18.77	3.57
POST								
INSP	10	-85	73	16.40	8.73	ns	27.62	9.32
EXP	10	-48	13	16.10	4.71	ns	14.79	9.83

TABLE 10

METHOD ERRORS - NASAL AIRWAY RESISTANCE
COMPARISON OF S(i) WITH A PREVIOUS STUDY

Variable	Current	Solow and Sandham 1988
ANT(L)		
INSP	37.91	33.3
EXP	38.48	24.3
ANT (R)		
INSP	35.90	41.5
EXP	18.77	32.7
POST		
INSP	27.62	18.9
EXP	14.79	13.6

4.2 POSTERO-ANTERIOR RADIOGRAPHS

All the following cephalometric procedures relating to postero-anterior and lateral radiographs are based on data recorded on a GTCO digitiser coupled to a Digipad 5 Screen. The errors that occur in this procedure are two-fold; the first relates to errors of point identification, the second to errors within the recording apparatus.

There have been few studies relating to the precision of digitisers (McWilliam and Welanders 1978; Eriksen and Solow 1991). It is obviously important that the linear validity of the digitising pad is checked, otherwise any given line will be recorded as having different lengths depending on where it is placed on the digitising pad. The accuracy of the digitising system used in this study was specified by the manufacturer as 0.1mm, and the resolution as 0.025mm. The accuracy of the digitiser was checked by the repeated recording of a set of points spread over the digitising area and the method errors found to be 0.08mm for the "x" axis and 0.14mm for the "y" axis.

Eriksen and Solow (1991) addressed the problems that digitisers may suffer from a further source of error, that of linearity due to the fact that electromagnetic fields are not homogenous over the entire surface of the screen due to the physical construction and location of the components of the system. They developed algorithms to assess and display the characteristics of digitisers, and suggested that inhomogenous fields can be electronically corrected by using the digitiser control unit.

It is generally accepted that direct digitising of the anatomical reference points from the radiographs is more reproducible than digitising from tracings (Richardson 1966, Houston 1982, Cohen 1984 and Sandham 1988) so direct digitising was utilised throughout the study.

Twenty postero-anterior films from the control sample had points defined, and digitised utilising the computer software developed for this study.

Four weeks later the process was repeated to enable comparisons to be made between the two sets of readings.

Results

The method errors for the P/A films are presented in Table 11.

No systematic differences were found, ($p > 0.05$). The method errors varied between 0.12 mm for MIR-C₂ and 0.51 mm for MSR-MSL.

The percentage errors varied between 1.3% for CNR-CNL and 9.3% for MSR-MSL.

TABLE 11

METHOD ERRORS P.A. RADIOGRAPHS
DISTRIBUTION OF THE DIFFERENCES BETWEEN DUPLICATE MEASUREMENTS

CL Sample

Variable	N	Min.	Max.	Mean Diff.	Standard error of Mean Difference	P	S(i)	S(i)%
CNR-CNM	20	-0.5	0.7	0.27	0.0440	ns	0.24	2.2
CNM-CNL	20	-1.3	1.0	0.37	0.0850	ns	0.37	5.1
MOR-MOL	20	-0.8	0.9	0.39	0.0670	ns	0.35	1.8
MSR-MSL	20	-1.0	1.5	0.51	0.1150	ns	0.51	9.3
CNR-CNL	20	-0.6	0.9	0.51	0.1180	ns	0.25	1.3
EMR-EML	20	-0.2	0.7	0.26	0.0430	ns	0.23	5.1
UMR-URL	20	-0.6	0.8	0.35	0.0366	ns	0.27	5.2
LMR-LML	20	-0.7	0.4	0.27	0.0384	ns	0.22	2.7
AGR-AGL	20	-0.7	1.5	0.44	0.0974	ns	0.43	7.6
MIR-MIL	20	-0.4	0.7	0.16	0.0456	ns	0.18	2.2
MOR-CNR	20	-0.9	1.1	0.31	0.0624	ns	0.29	8.8
MOL-CNL	20	-0.5	1.0	0.42	0.0555	ns	0.34	8.4
CNR-EMR	20	-0.5	0.7	0.25	0.0438	ns	0.22	6.2
CNL-EML	20	-1.0	1.1	0.35	0.0534	ns	0.26	8.7
EMR-UMR	20	-0.8	1.3	0.45	0.0783	ns	0.40	2.0
EML-UML	20	-0.5	0.6	0.21	0.0355	ns	0.19	4.2
UML-MIR	20	-0.8	1.3	0.38	0.0523	ns	0.31	6.6
MIL-UML	20	-0.8	1.1	0.35	0.0738	ns	0.33	7.5
LOR-UMR	20	-0.6	0.3	0.47	0.0941	ns	0.44	4.9
LOL-UML	20	-1.2	1.1	0.54	0.0883	ns	0.47	6.1
MIR-C ₂	20	-0.4	0.5	0.09	0.0328	ns	0.12	2.9
C ₂ -MIL	20	-0.7	0.4	0.13	0.0428	ns	0.14	3.9
NASDIM	20	-0.5	0.7	0.43	0.0664	ns	0.37	3.1
MAXDIM	20	-0.5	0.7	0.37	0.0792	ns	0.36	5.4
MANDDIM	20	-0.6	1.1	0.37	0.0821	ns	0.36	6.8

4.3 HEAD POSTURE ANALYSIS

In order to assess the error of head posture measurement, ten subjects (6 female, 4 male) aged between 10 and 15 years had duplicate lateral skull radiographs taken. All these subjects were from the anomaly sample.

These duplicate radiographs were exposed only if the original was deemed unsatisfactory due to missed cranial structures, overexposure of soft tissue or incorrect bite registration. The elapsed time between the original and duplicate radiographs was a minimum of one hour, and any duplicates were requested only after scrutiny of the originals by the author. Hence, any unnecessary exposure to ionising radiation was avoided.

The same Superintendent Radiographer exposed both the original and the duplicate films utilising the same cephalometric machine and the same type and manufacture of film on each occasion.

The sample was accumulated over a period of six months. The method for positioning the head in its natural position was that developed by Solow and Tallgren (1971), as described in the Methodology section.

The relevant cephalometric points on the original and duplicate radiographs were digitised utilising computer software developed specifically for this project.

The variables recorded are from Table 5, ie

- NSL/VER
- NSL/OPT
- NSL/CVT
- OPT/HOR
- CVT/HOR
- OPT/CVT

Results

The results of the method error study for head posture are given in Table 12.

They show no systematic difference between the two series, ($p > 0.05$). The method error ranged between 1.08 degrees for OPT/CVT to 2.23 degrees for NSL/CVT.

These results are comparable with those of previous studies (see Table 13).

The error percentages ranged from 2.3% for NSL/CVT to 12.4% for NSL/VER and OPT/HOR.

METHOD ERRORS - POSTURAL ANGLES
DISTRIBUTION OF DIFFERENCES BETWEEN
FIRST AND SECOND RECORDING OF POSTURAL ANGLES

Anomaly Sample

Variable	N	Min.	Max.	Mean Diff.	Standard error of Mean Difference	P	S(i)	S(i)%
NSL/VER	10	-3.6	5.0	0.13	0.46	ns	1.61	12.4
NSL/OPT	10	-4.9	4.6	0.33	0.54	ns	1.73	10.3
NSL/CVT	10	-6.7	5.2	0.07	0.74	ns	2.23	2.3
OPT/HOR	10	-3.1	2.8	0.26	0.35	ns	1.24	12.4
CVT/HOR	10	-7.0	2.8	0.01	0.61	ns	1.94	10.1
OPT/CVT	10	-1.7	3.9	0.26	0.35	ns	1.08	9.4

TABLE 13

METHOD ERRORS - POSTURAL ANGLES
COMPARISON OF S(i) WITH PREVIOUS STUDIES

	SANDHAM 1988	SOLOW & SIERSBAEK- NIELSEN 1982	PRESENT
NSL/VER	3.20	2.25	1.61
NSL/OPT	2.56	3.39	1.73
NSL/CVT	2.44	3.37	2.23
OPT/HOR	3.84	3.07	1.24
CVT/HOR	3.25	3.09	1.94
OPT/CVT	0.97	0.90	1.08

4.4 CEPHALOMETRIC VARIABLES

For assessment of method errors, twenty lateral films from the control sample had cephalometric points defined then digitised. Four weeks later this procedure was repeated to enable comparison of the measurements obtained for the same linear and angular variables.

The differences between the measurements on the initial and secondary tracings were calculated and a statistical analysis performed.

Results

The method errors are reported in Table 14.

No significant differences ($p > 0.05$) were found. The method errors between the first and second set of records for angular measurements ranged between 0.31 degrees for ss-n-pg to 1.53 degrees for IL_S/NL . The method error for linear measurements varied between 0.33 mm for Int to n-ss and 1.05 mm for n_S -prn.

The percentage error for angular values ranged between 4.37% for NSL/ML and 6.58% for n-s-ar. The percentage error for linear values ranged between 2.08% for sp-is and 8.62% for n-s.

TABLE 14

METHOD ERRORS - CEPHALOMETRICS
DISTRIBUTION OF THE DIFFERENCES BETWEEN THE DUPLICATE MEASUREMENTS

CL Sample

Variable	N	Min.	Max.	Mean Diff.	Standard error of Mean Difference	P	S(i)	S(i)%
n-s	20	-0.40	1.10	0.44	0.153	ns	0.449	8.62
n-sp	20	-1.10	0.60	-0.18	0.195	ns	0.453	5.40
n-gn	20	-2.10	0.90	-0.11	0.339	ns	0.749	4.87
s-ba	20	-1.60	1.40	-0.13	0.272	ns	0.599	4.67
s-ar	20	-1.40	1.10	-0.15	0.296	ns	0.637	4.63
s-pm	20	-2.10	2.50	0.16	0.437	ns	0.933	4.56
s-tg	20	-0.90	1.10	0.24	0.187	ns	0.431	5.32
sp-gn	20	0.00	1.00	0.10	0.187	ns	0.446	4.40
ar-tgo	20	-0.90	1.00	0.11	0.213	ns	0.458	4.64
sp-pm	20	-1.80	2.80	0.49	0.393	ns	0.675	3.22
ss-pm	20	-1.20	2.30	0.15	0.356	ns	0.673	3.57
pgn-cd	20	-2.10	0.70	-0.33	0.281	ns	0.639	5.18
pg-tgo	20	-1.00	1.40	0.23	0.241	ns	0.552	5.25
sp-is	20	-1.10	1.10	0.14	0.199	ns	0.434	2.08
ii-gn	20	-1.10	0.90	-0.17	0.207	ns	0.455	4.83

TABLE 14 (cont)
DISTRIBUTION OF THE DIFFERENCES BETWEEN THE DUPLICATE MEASUREMENTS

CL Sample	Variable	N	Min.	Max.	Mean Diff.	Standard error of Mean Difference	P	S(i)	S(i)%
	NL/VER	20	-2.30	1.20	-0.52	0.369	ns	0.529	5.54
	NSL/OPT	20	-3.90	2.90	0.07	0.563	ns	1.194	4.50
	NSL/CVT	20	-2.00	1.60	-0.03	0.347	ns	0.737	4.50
	NL/OPT	20	-2.00	3.20	0.51	0.505	ns	1.130	5.00
	NL/CVT	20	-1.50	3.70	0.44	0.471	ns	1.047	4.93
	OPT/HOR	20	-2.40	1.70	-0.43	0.445	ns	1.128	6.43
	CVT/HOR	20	-1.60	1.00	-0.36	0.274	ns	0.634	5.37
	FH/VER	20	-1.40	1.70	0.10	0.345	ns	0.736	4.54
	FH/OPT	20	-2.50	3.00	0.53	0.463	ns	1.053	5.18
	FH/CVT	20	-0.80	1.80	0.43	0.287	ns	0.709	6.12
	pm-ad ₁	20	-2.20	1.80	0.02	0.380	ns	0.806	4.49
	pm-ad ₂	20	-2.50	1.90	0.27	0.467	ns	1.009	4.66
	pm-ad ₃	20	-1.90	2.30	0.21	0.406	ns	0.884	4.75
	tu-ad ₃	20	-0.90	1.40	0.05	0.227	ns	0.482	4.52
	n _s -sn	20	-2.90	2.10	-0.22	0.487	ns	1.045	4.60
	n _s -prn	20	-2.80	1.70	-0.31	0.484	ns	1.046	4.67
	Int to n-ss	20	-0.80	0.60	-0.06	0.153	ns	0.327	4.57

TABLE 14 (cont)

DISTRIBUTION OF THE DIFFERENCES BETWEEN THE DUPLICATE MEASUREMENTS

CL Sample	Variable	N	Min.	Max.	Mean Diff.	Standard error of Mean Difference	P	S(i)	S(i)%
	s-n _s -unt sto to NL	20	-2.80	3.30	-0.08	0.693	ns	1.475	4.53
	s-n _s -ss _s	20	-0.80	1.30	0.05	0.213	ns	0.452	4.52
	sn to Int-l _s	20	-2.10	1.60	0.03	0.332	ns	0.705	4.49
	ls to NCL	20	-1.10	0.90	-0.19	0.199	ns	0.360	3.26
	sto to ML	20	-0.30	1.20	0.32	0.173	ns	0.434	6.19
	s-n _s -sm _s	20	-2.40	1.10	-0.24	0.296	ns	0.650	4.82
	sm _s to li-pg _s	20	-1.10	1.30	0.03	0.255	ns	0.542	4.51
	li to NCL	20	-0.80	0.70	-0.01	0.145	ns	0.330	5.19
	ss _s -n _s -sm _s	20	-2.00	0.90	-0.20	0.259	ns	0.342	4.18
	sto to OL _s	20	-0.80	1.20	-0.07	0.207	ns	0.442	4.55
	S-n _s -pg _s	20	-1.50	1.30	-0.01	0.280	ns	0.374	7.98
	NFL/NCL	20	-1.00	2.00	0.15	0.301	ns	0.647	4.63
		20	-0.70	1.00	0.10	0.194	ns	0.418	4.62

RESULTS

5.1 DIFFERENCES BETWEEN THE CONTROL SAMPLE AND THE ANOMALY SAMPLE BEFORE TREATMENT

A) Nasal Airway Resistance

Nasal airway resistance was recorded bilateral for the total nasopharyngeal airway in all subjects, as well as unilaterally for each nasal half. This enabled a general comparison of total upper airway resistance to be made, and also provided a breakdown of the resistance of each nasal component.

Comparisons of the mean rhinomanometric variables between males and females in the control groups revealed only 3 statistically significant differences ($p < 0.05$) out of a total of 18 separate comparisons (see Tables 15 and 16).

Similar comparisons between males and females in the anomaly sample before treatment revealed no statistically significant differences between the means (Tables 17 and 18).

It was concluded that for the purposes of comparing the rhinomanometric variables between the control sample and the anomaly sample before treatment, it would be reasonable to combine the data for the sexes.

Total upper airway resistance

An overview of all the rhinomanometric values of the control sample and anomaly sample before treatment including the laminar and turbulent components is shown in Table 21. There were no significant differences for nasal airway resistance. If one however turns to the laminar and turbulent components of the comparison of total airway resistance expressed in pascals/cc/second $\times 10^3$ (Table 22), there is a statistically significant difference between the mean laminar flow on inspiration (150.27 for the control sample against 196.84 for the anomaly sample before treatment), for the laminar

component of expiration (146.45 for the control against 186.53 for the anomaly sample before treatment), as well as in the turbulent component (353.47 for the control sample compared with 714.85 for the anomaly sample before treatment on inspiration) and for the turbulent component of expiration (352.76 for the control sample compared with 710.56 for the anomaly sample before treatment).

Unilateral nasal respiratory resistance

The unilateral analysis of nasal resistance in the control group measured against the anomaly sample before treatment showed statistically significant differences at the $p < 0.001$ level, for both inspiration and expiration, measured on both right and left sides. The values for the anomaly sample before treatment were considerably higher than for the control sample (see Table 21). The mean values in pascals/cc/second $\times 10^3$ for the control sample ranged from ANT L EXP of 603.52 to ANT R EXP of 632.87. The mean values in the anomaly sample before treatment ranged from 819.48 for ANT R EXP to 827.98 for ANT R INSP.

For laminar and turbulent flow statistically significant differences were found between controls and anomaly samples (Table 22). The mean laminar flow values for the control sample ranged from 190.04 for the laminar L EXP to 203.86 for the laminar R EXP whereas those for the anomaly sample before treatment ranged from 239.43 for the laminar L EXP to 267.27 for the laminar R INSP, ($p < 0.001$).

The range of the turbulent flow was from 845.83 for the L EXP value to 916.64 for the R INSP value in the controls, as compared with a range of 1233.87 for R EXP to 1328.92 for L INSP in the anomaly sample before treatment.

TABLE 15

NASAL AIRWAY RESISTANCE - CONTROL SAMPLE
N A R (pascals/cc/sec x 10³) at 150 pascals

Variable	Number*	male			female								
		Min	Max	Mean	S.D.	Number*	Min	Max	Mean	S.D.	female Diff.	t	p
<u>ANT L</u>													
INSP	9	265	721	524.22	143.59	16	333	1070	670.87	194.79	+146.65	-1.97	0.061
EXP	9	247	744	504.66	144.67	16	303	1084	659.13	203.32	+154.47	-2.00	0.057
<u>ANT R</u>													
INSP	9	452	868	606.78	132.04	14	366	1169	648.21	219.71	+41.43	-0.51	0.617
EXP	9	452	882	596.33	145.71	14	348	1196	656.38	230.79	+60.05	-0.69	0.496
<u>POST</u>													
INSP	11	274	702	437	129.94	22	250	683	429.14	101.95	- 7.86	0.19	0.852
EXP	11	264	628	410	118.95	22	242	699	415.29	111.67	+ 5.29	-0.12	0.902

* The number varies because certain subjects were unable to complete all tests

TABLE 16

NASAL AIRWAY RESISTANCE - CONTROL SAMPLE
Laminar and Turbulent flow coefficients K₁/K₂ (pascals/cc/sec x 10⁴) at 150 pascals

Variable	male					female					t	p	
	Number*	Min	Max	Mean	S.D.	Number*	Min	Max	Mean	S.D.			Diff.
K ₁ L INS	9	100	280	186.00	54.87	16	110	412	198.13	75.39	+ 12.13	-0.40	0.641
K ₁ L EXP	9	113	279	184.88	49.53	16	115	317	192.80	62.87	+ 7.92	-0.31	0.702
K ₁ R INSP	9	109	270	177.78	45.61	14	124	334	208.92	50.00	+ 31.14	-1.47	0.104
K ₁ R EXP	9	107	265	178.44	44.17	14	126	366	222.92	65.59	+ 44.48	-1.75	0.042
K ₁ P INS	11	117	204	158.44	28.58	22	92	294	146.76	45.28	- 11.68	0.71	0.414
K ₁ P EXP	11	100	192	149.89	30.77	22	91	314	145.05	46.24	- 4.84	0.29	0.742
K ₂ L INS	9	792	1227	1061.25	167.23	16	312	1204	762.75	321.72	-298.50	2.45	0.038
K ₂ L EXP	9	789	1212	1036.38	171.05	16	260	1151	744.20	323.67	-292.18	2.37	0.034
K ₂ R INS	9	660	1306	1029.78	240.07	14	336	1219	838.31	325.06	-191.47	1.50	0.142
K ₂ R EXP	9	656	1302	1001.33	226.20	14	363	1191	831.77	322.94	-169.56	1.36	0.131
K ₂ P INS	11	225	656	366.36	117.19	22	115	710	348.71	133.52	- 17.65	0.41	0.641
K ₂ P EXP	11	188	658	354.00	125.42	22	124	653	352.14	125.66	- 1.86	0.04	0.943

TABLE 17

NASAL AIRWAY RESISTANCE - ANOMALY SAMPLE BEFORE TREATMENT
N A R (pascals/cc/sec x 10³) at 150 pascals

Variable	Number*	Min	Max	Mean	S.D.	Number*	Min	Max	Mean	female S.D.	Diff.	t	p
<u>ANT L</u>													
INSP	27	473	1602	828.44	246.12	32	460	1746	827.44	220.76	-1.00	0.02	0.987
EXP	27	475	1590	828.36	255.80	32	425	1668	823.22	207.00	-5.14	0.08	0.933
<u>ANT R</u>													
INSP	25	477	1219	797.12	216.00	30	494	1565	853.70	237.67	+56.58	-0.92	0.364
EXP	25	438	1312	808.33	230.75	30	461	1514	828.40	231.29	+20.07	-0.32	0.752
<u>POST</u>													
INSP	27	302	971	493.03	172.42	31	324	880	481.29	124.01	-11.74	0.30	0.765
EXP	27	293	900	467.33	166.22	31	306	806	453.55	115.58	-13.78	0.37	0.712

* The number varies because certain subjects were unable to complete all tests

TABLE 18

NASAL AIRWAY RESISTANCE - ANOMALY SAMPLE BEFORE TREATMENT
Laminar and Turbulent flow coefficients K₁/K₂ (pascal/cc/sec x 10⁴) at 150 pascals

Variable	male			female			t	p
	Number*	Min	Max	Mean	S.D.	Diff.		
K ₁ L INS	27	120	412	238.60	84.32			
K ₁ L EXP	27	110	400	226.17	81.99	+ 35.40	-1.48	0.146
K ₁ R INSP	25	110	448	243.57	87.21			
K ₁ R EXP	25	108	423	235.44	82.46	+ 25.05	-1.20	0.234
K ₁ P INS	27	120	484	196.00	92.13	+ 48.48	-1.35	0.184
K ₁ P EXP	27	97	478	187.55	98.67	+ 43.70	-1.26	0.215
K ₂ L INS	27	563	1818	1331.33	329.92			
K ₂ L EXP	27	547	1934	1318.82	337.99	+ 1.48	-0.06	0.950
K ₂ R INS	25	374	1822	1220.95	413.59	- 1.79	0.08	0.940
K ₂ EXP	25	397	1770	1164.16	395.57	- 4.29	0.05	0.963
K ₂ P INS	27	336	1571	732.46	303.15	- 79.38	0.86	0.395
K ₂ P EXP	27	322	1512	741.65	342.94	+161.83	-1.38	0.177
						+143.28	-1.21	0.233
						- 30.53	0.34	0.736
						- 53.27	0.57	0.574

* The numbers vary because certain subjects were unable to complete all tests

TABLE 19

NASAL AIRWAY RESISTANCE - ANOMALY SAMPLE AFTER TREATMENT
N A R (pascals/cc/sec x 10³) at 150 pascals

Variable	male Number*	Min	Max	Mean	S.D.	Number*	Min	Max	female Mean	S.D.	Diff.	t	p
<u>ANT L</u>													
INSP	27	411	996	646.70	153.98	32	449	966	665.39	129.95	-18.69	-0.50	0.618
EXP	27	410	957	633.52	148.95	32	428	949	640.00	133.00	-6.48	-0.17	0.864
<u>ANT R</u>													
INSP	25	441	827	610.39	102.28	30	356	1037	675.24	171.71	-64.85	-1.57	0.123
EXP	25	412	776	593.61	95.26	30	335	1069	665.56	187.18	-71.95	-1.66	0.105
<u>POST</u>													
INSP	27	289	590	365.93	57.56	31	249	883	421.89	133.32	-55.96	-2.01	0.049
EXP	27	279	511	344.78	51.04	31	222	851	402.10	131.53	-57.32	-2.12	0.039

* The number varies because certain subjects were unable to complete all tests

TABLE 20

NASAL AIRWAY RESISTANCE - ANOMALY SAMPLE AFTER TREATMENT
Laminar and Turbulent flow coefficients K₁/K₂ (pascal/cc/sec x 10³) at 150 pascals

Variable	Number*	male			Number*	Min	Max	Mean	S.D.	Diff.	t	p	
		Min	Max	Mean									
K ₁ L INS	27	100	295	161.04	49.39	32	126	268	192.04	36.68	-31.00	-2.57	0.013
K ₁ L EXP	27	93	244	149.18	43.35	32	125	246	181.63	35.36	-32.45	-2.89	0.006
K ₁ R INS	25	113	372	176.09	67.91	30	112	267	198.35	43.17	-22.26	-1.25	0.221
K ₁ R EXP	25	105	368	167.16	61.19	30	121	305	190.75	46.91	-23.59	-1.36	0.183
K ₁ P INS	27	62	178	116.75	30.47	31	70	259	144.22	50.79	-27.47	-2.15	0.037
K ₁ P EXP	27	56	180	113.65	32.71	31	56	220	133.78	44.89	-20.13	-1.70	0.097
K ₂ L INS	27	471	1841	998.00	307.64	32	297	1636	1000.04	328.21	- 2.04	-0.02	0.982
K ₂ L EXP	27	512	1773	975.81	293.37	32	287	1660	957.04	328.24	18.77	0.20	0.839
K ₂ R INS	25	276	1208	903.94	289.22	30	374	1854	991.89	401.57	-87.95	-0.76	0.452
K ₂ R EXP	25	273	1268	845.35	301.95	30	343	1752	947.74	370.07	102.39	-0.90	0.373
K ₂ P INS	27	132	900	437.95	165.54	31	137	1060	512.85	207.66	- 74.90	-1.35	0.183
K ₂ P EXP	27	116	846	451.35	177.41	31	130	1040	501.19	205.08	-49.84	-0.87	0.391

*The numbers vary because certain subjects were unable to complete all tests

TABLE 21

NASAL AIRWAY RESISTANCE: COMPARISON OF CONTROL AND ANOMALY SAMPLES BEFORE TREATMENT
N A R (pascals/cc/sec $\times 10^3$) at 150 pascals

Variable	Control Sample Number*	Min	Max	Mean	S.D.	Number*	Min	Max	Mean	S.D.	Diff.	t	p
<u>ANT L</u>													
INSP	25	265	1070	618.08	189.08	57	460	1746	827.89	230.66	209.81	4.01	0.001
EXP	25	247	1084	603.52	196.31	57	425	1668	825.47	227.54	221.91	4.23	0.001
<u>ANT R</u>													
INSP	23	366	1169	632.00	187.86	55	477	1565	827.98	227.77	195.98	3.64	0.001
EXP	23	348	1196	632.87	200.24	55	438	1514	819.48	229.08	186.61	3.39	0.001
<u>POST</u>													
INSP	33	250	702	431.84	110.30	58	302	971	486.76	147.27	54.92	1.84	0.069
EXP	33	242	699	413.47	112.32	58	293	900	459.08	140.29	46.50	1.61	0.111

* The numbers vary because certain subjects were unable to complete all tests

TABLE 22

NASAL AIRWAY RESISTANCE: COMPARISON OF CONTROL AND ANOMALY SAMPLES BEFORE TREATMENT
Laminar and Turbulent flow coefficients K1/K2 (pascal/cc/sec x 10³) at 150 pascals

Variable	Control Sample Number*	Min	Max	Mean	S.D.	Number*	Min	Max	Mean	S.D.	Diff.	t	p
<u>ANT L</u>													
INSP K ₁	25	100	412	194.08	68.25	59	120	593	257.30	88.08	63.22	-3.11	0.003
EXP K ₁	25	113	317	190.04	57.54	59	110	410	239.43	74.52	49.39	-2.82	0.006
<u>ANT R</u>													
INSP K ₁	23	109	334	195.57	49.56	55	110	876	267.27	121.49	68.70	-2.60	0.012
EXP K ₁	23	107	366	203.86	60.46	55	108	848	256.80	117.09	52.94	-1.95	0.056
<u>POST</u>													
INSP K ₁	33	92	294	150.27	40.85	58	102	484	196.84	82.21	46.57	-2.89	0.005
EXP K ₁	33	91	314	146.45	41.88	58	97	478	186.53	82.73	40.08	-2.51	0.014
<u>ANT L</u>													
INSP K ₂	25	312	1227	862.25	310.93	59	452	1933	1328.92	314.61	466.67	-5.96	0.001
EXP K ₂	25	260	1212	845.83	310.19	59	447	1934	1276.59	315.42	430.76	-5.40	0.001
<u>ANT R</u>													
INSP K ₂	23	336	1306	916.64	302.68	55	374	1907	1297.61	365.70	380.97	-4.13	0.001
EXP K ₂	23	363	1302	901.14	293.89	55	397	1887	1233.87	360.98	332.73	-3.66	0.001
<u>POST</u>													
INSP K ₂	33	115	710	353.47	126.58	58	177	1571	714.85	318.01	361.38	-6.13	0.001
EXP K ₂	33	124	658	352.76	123.61	58	176	1512	710.56	319.51	357.80	-6.12	0.001

*The numbers vary because certain subjects were unable to complete all tests

B) Transverse Craniofacial Dimensions

The measurements of the transverse dimensions on the postero-anterior cephalometric radiographs were compared between the sexes in the control sample, and the anomaly sample before treatment (Tables 23,24,25).

In the control sample there was a statistically significant difference ($p=0.035$) only between EMR-EML, the width of the maxillary base.

In the anomaly sample before treatment there were statistically significant differences in nasal width (L), CNM-CNL ($p=0.010$), maxillary base width EMR-EML ($p=0.004$) and lower molar width LMR-LML ($p=0.033$).

In practical terms it appeared reasonable to combine the male and female values in the control sample and the anomaly sample before treatment because the differences between the mean values were so small.

In the anomaly sample after treatment, more variables showed statistically significant differences between the mean values for males and females: EMR-EML ($p=0.007$) and UMR-UML ($p=0.035$), LMR-LML ($p=0.032$), MIR-MIL ($p=0.047$) and MIR-C₂ ($p=0.030$), the male mean value being consistently larger than the female in all cases.

Separate consideration will therefore be given to each sex in a subsequent analysis. Consideration will also be given to the variable EMR-EML, which was found to be statistically significantly greater in males in all three samples. The mean transverse dimensions are shown in Table 26. Comparison between the control sample and the anomaly sample before treatment showed a number of statistically significant differences at the $p<0.001$ and $p<0.05$ levels, particularly in respect to the horizontal values, all of which were smaller in the treatment (see Figure35).

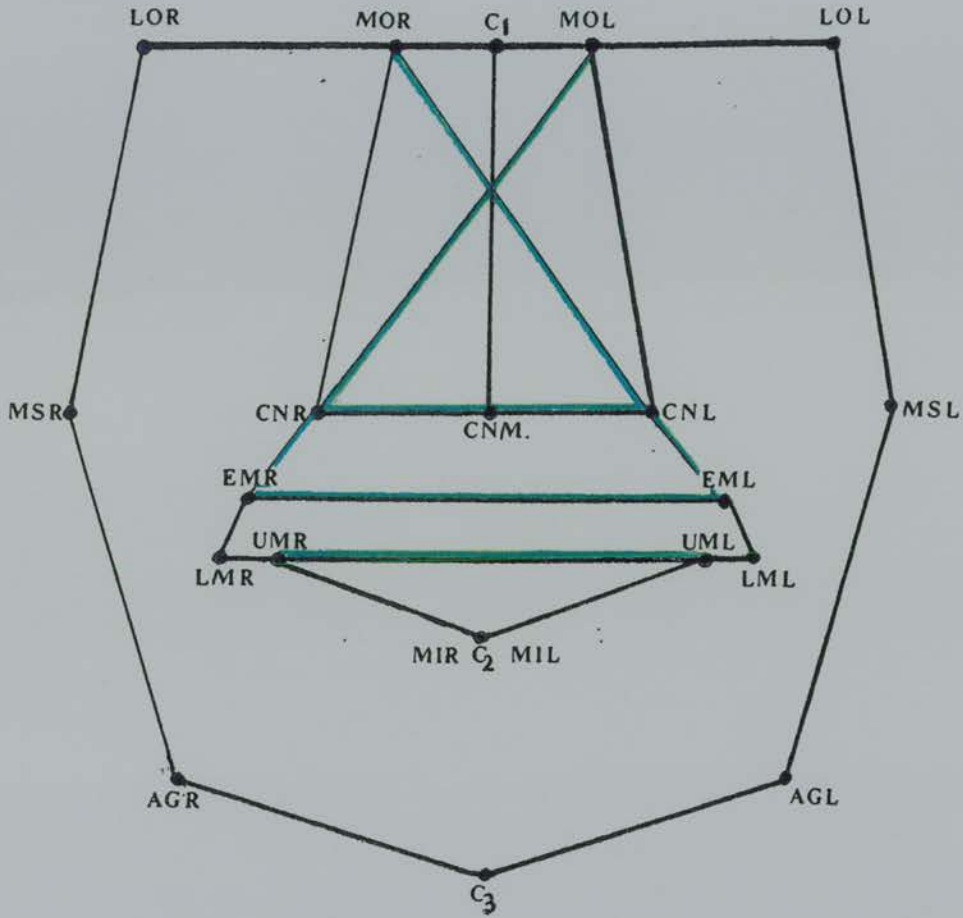
The measurement CNR-CNL showed a mean value of 29.64mm in the control sample and 28.00mm in the anomaly sample before treatment ($p < 0.005$), showing that the width of the anterior nasal floor was significantly smaller in the anomaly sample before treatment than in the control group by some 6%.

The measurement EMR-EML in the control sample had a mean value of 63.87mm, compared with 60.99mm in the anomaly sample before treatment - the difference was statistically significant at the $p < 0.001$ level. The measurement UMR-UML had a mean value of 61.21mm in the control sample and 53.86mm in the anomaly sample before treatment ($p < 0.001$). Hence the maxillary base width and the width between the lateral points on the maxillary molars were significantly narrower in the anomaly sample before treatment than in the control sample.

The variable NAS DIM, which comprises a line from MOR to CNL and a line from MOL to CNR, showed significant differences between the two samples at the $p < 0.001$ level, as did the variable MAX DIM which comprises a line from MOR to EML and a line from MOL to EMR, all measured in mm.

The mean values for NAS DIM were 70.79 for the control sample against 66.99 for the anomaly before sample. The values for MAX DIM were 82.31 for the control and 78.31 for the anomaly before sample.

POSTERO ANTERIOR CEPHALOMETRIC RESULTS



SIGNIFICANT DIFFERENCES

CONTROL SAMPLE/ANOMALY SAMPLE BEFORE TREATMENT

Fig. 35

TABLE 23

TRANSVERSE CEPHALOMETRIC DIMENSIONS IN MM - CONTROL SAMPLE

Variable	male					female					Diff.	t	p
	Number	Min	Max	Mean	S.D.	Number	Min	Max	Mean	S.D.			
CNR-CNM	12	13.9	20.6	16.45	1.95	24	14.3	19.5	16.66	1.53	+0.21	-0.35	0.732
CNM-CNL	12	14.2	19.3	16.22	1.71	24	14.0	18.9	16.39	1.57	+0.17	-0.31	0.760
CNR-CNL	12	26.3	34.9	29.63	3.05	24	25.80	34.60	29.65	2.33	+0.02	-0.01	0.993
EMR-EML	12	57.9	69.5	65.63	2.98	24	54.70	69.80	62.99	3.57	-2.64	2.20	0.041
UMR-UML	12	54.4	64.7	62.03	2.94	24	53.80	67.00	60.80	3.35	-1.23	1.08	0.294
LMR-LML	12	49.0	65.4	58.78	4.30	24	50.80	63.60	56.47	3.39	-2.31	1.77	0.091
MIR-MIL	12	00.0	2.60	0.43	0.89	24	0.00	4.10	0.46	1.09	+0.03	-0.10	0.922
MIR-C2	12	00.0	1.30	0.23	0.44	24	0.00	2.60	0.27	0.63	+0.04	-0.20	0.840
C2-MIL	12	00.0	1.30	0.22	0.44	24	0.00	1.50	0.21	0.47	-0.01	0.05	0.962
NAS-DIM	12	65.5	86.00	72.00	5.75	24	62.00	84.00	70.18	5.65	-0.82	-0.91	0.371
MAX-DIM	12	67.2	95.10	83.43	5.69	24	75.10	91.10	81.75	4.82	-1.68	-0.93	0.363

TABLE 24

TRANSVERSE CEPHALOMETRIC DIMENSIONS IN MM - ANOMALY SAMPLE BEFORE TREATMENT

Variable	Number	male				female				Diff.	t	p	
		Min	Max	Mean	S.D.	Number	Min	Max	Mean				S.D.
CNR-CNM	31	12.8	19.5	16.23	1.76	41	12.9	19.9	15.48	1.65	-0.75	1.87	0.070
CNM-CNL	31	11.9	20.0	15.97	1.88	41	12.5	18.1	14.94	1.46	-1.03	2.64	0.010
CNR-CNL	31	23.1	35.0	28.75	2.98	41	20.2	33.0	27.44	2.83	-1.31	1.90	0.062
EMR-EML	31	53.6	68.8	62.65	4.19	41	51.5	67.8	59.74	3.93	-0.91	3.02	0.004
UMR-UML	31	44.2	66.2	54.93	4.99	41	44.2	60.2	53.05	4.12	-1.88	1.75	0.090
LMR-LML	31	48.9	70.7	59.70	4.30	41	44.4	67.24	57.51	4.17	-2.19	2.18	0.033
MIR-MIL	31	0.0	5.3	0.58	1.38	41	0.00	2.3	0.22	0.51	-0.36	1.55	0.127
MIR-C2	31	0.0	2.4	0.27	0.58	41	0.00	1.2	0.13	0.23	-0.14	1.40	0.170
C2-MIL	31	0.0	3.4	0.34	0.81	41	0.00	1.4	0.12	0.29	-0.22	1.63	0.120
NAS-DIM	31	58.3	79.0	67.67	4.59	41	55.5	79.5	66.48	5.71	-1.19	-0.95	0.348
MAX-DIM	31	68.4	86.8	78.13	4.67	41	69.1	89.5	78.45	5.90	+0.32	0.25	0.803

TABLE 25

TRANSVERSE CEPHALOMETRIC DIMENSIONS IN MM - ANOMALY SAMPLE AFTER TREATMENT

Variable	Number	Min	Max	male Mean	S.D.	Number	Min	Max	female Mean	S.D.	Diff.	t	P
CNR-CNM	31	14.7	21.5	17.41	1.58	41	14.6	20.2	16.75	1.41	-0.66	1.87	0.065
CNM-CNL	31	14.4	20.8	17.22	1.48	41	11.6	20.2	16.45	1.82	-0.77	1.92	0.058
CNR-CNL	31	24.5	37.3	31.51	2.72	41	25.0	37.5	30.55	2.91	-0.96	1.42	0.159
EMR-EML	31	56.9	76.6	64.60	4.51	41	54.4	71.0	62.03	3.29	-2.57	2.80	0.007
UMR-UML	31	51.7	72.5	62.93	5.44	41	50.2	66.0	60.64	3.57	-2.29	2.15	0.035
LMR-LML	31	49.9	70.8	60.05	4.29	41	44.5	68.1	57.86	4.11	-2.19	2.19	0.032
MIR-MIL	31	2.7	7.7	5.54	1.49	41	2.3	8.2	4.82	1.51	-0.72	2.03	0.047
MIR-C2	31	1.1	3.9	2.69	0.76	41	1.1	3.9	2.29	0.78	-0.40	2.21	0.030
C2-MIL	31	1.5	4.2	2.85	0.77	41	1.1	4.3	2.54	0.78	-0.31	1.69	0.096
NAS-DIM	31	58.3	82.5	70.18	5.11	41	56.0	82.0	68.97	6.18	-1.21	0.89	0.380
MAX-DIM	31	68.0	90.3	78.28	5.06	41	69.0	90.0	78.87	5.42	+0.59	0.47	0.640

TABLE 26

COMPARISON OF TRANSVERSE CEPHALOMETRIC DIMENSIONS IN MM
BETWEEN CONTROL AND ANOMALY SAMPLES

Variable	Number	Control Sample		Mean	S.D.	Number	Min	Max	Anomaly Before Sample		Diff.	t	p
		Min	Max						Mean	S.D.			
CNR-CNM	36	13.90	20.60	16.55	1.74	72	12.80	19.90	15.85	1.70	-0.70	2.27	0.025
CNM-CNL	36	14.00	19.30	16.31	1.77	72	11.90	20.00	15.46	1.670	-0.85	2.78	0006
CNR-CNL	36	25.80	34.90	29.64	2.275	72	20.20	35.00	28.00	2.948	-1.64	2.85	0.005
EMR-EML	36	54.70	69.80	63.87	3.570	72	51.50	68.80	60.99	4.271	-2.88	3.47	0.001
UMR-UML	36	53.80	67.00	61.21	3.232	72	44.20	66.20	53.86	4.579	-7.35	8.60	0.001
LMR-LML	36	49.00	65.40	57.24	3.822	72	44.40	70.70	58.45	4.340	1.21	-1.42	0.157
MIR-MIL	36	0.00	4.10	0.45	1.012	72	0.000	5.30	0.38	0.991	-0.07	0.37	0.713
MIR-C2	36	0.00	2.60	0.25	0.570	72	0.00	2.40	0.19	0.418	-0.06	0.66	0.510
C2-MIL	36	0.00	1.50	0.21	0.452	72	0.00	3.40	0.21	0.577	0.00	-0.01	0.990
NAS-DIM	36	62.00	86.00	70.79	5.668	72	55.50	79.50	66.99	5.258	-3.80	-3.45	0.001
MAX-DIM	36	67.20	95.10	82.31	5.108	72	68.40	89.50	78.31	5.373	4.00	-3.71	0.001

C) Head Posture

The mean head posture measurements of the control sample in males and females (Table 27, 28, 29) showed no statistically significant differences. In the anomaly sample before treatment, statistically significant differences between the sexes were found for NL/VER and CVT/HOR ($p < 0.05$) and in the same sample after treatment the mean values of NSL/CVT and CVT/HOR showed statistically significant differences between males and females ($p < 0.05$). In all the instances the mean values for males exceeded those for females.

It was concluded however that, for the purposes of this investigation, it would be reasonable to combine the data for the sexes in each of the three groups.

The comparison of head posture variables in the control sample with those in the anomaly sample before treatment (see Table 30) showed no statistically significant differences:-

However, the variables associated with a backwards inclination of the head showed a definite trend, with the larger values in the anomaly sample before treatment. Hence NSL/VER showed a mean value of 95.33° in the control group compared with a mean value of 96.69° in the anomaly group before treatment. The mean values of NSL/OPT were 98.85° in the control group and 101.41° in the anomaly group before treatment. The corresponding values of NSL/CVT were 103.85° and 105.47° respectively.

The values NSL/CVT and NSL/OPT have particular relevance as these were the values used in previous studies (Siersbaek-Nielsen and Solow 1982; Solow et al 1984; Sandham 1987) to determine craniocervical angulation, as they lie remote from the areas directly affected by the mechanical aspects of RME.

TABLE 27

HEAD POSTURE IN DEGREES - CONTROL SAMPLE

Value	male n = 12			female n = 24									
	Number	Min	Max	Mean	S.D.	Number	Min	Max	Mean	S.D.	Diff.	t	p
NSL/VER	12	82.6	110.0	94.68	8.24	24	85.8	107.3	95.67	6.29	0.99	-0.40	0.690
NL/VER	12	80.5	104.0	88.17	7.31	24	72.0	102.5	87.44	7.43	0.73	0.28	0.780
NSL/OPT	12	80.8	108.2	97.45	7.70	24	81.4	115.9	99.55	9.53	2.10	-0.66	0.513
NSL/CVT	12	86.3	115.3	100.47	7.34	24	89.3	123.5	105.55	9.16	5.08	-1.67	0.100
NL/OPT	12	70.0	100.5	91.28	9.00	24	73.8	111.1	91.62	9.49	0.34	-0.10	0.920
NL/CVT	12	75.4	104.6	94.28	7.78	24	81.7	118.6	97.62	9.24	3.34	-1.07	0.290
OPT/HOR	12	74.7	100.9	87.23	7.44	24	68.5	102.9	86.11	8.64	-1.12	0.38	0.700
CVT/HOR	12	75.4	95.4	84.18	6.28	24	64.7	94.0	80.11	7.98	-4.07	1.54	0.130
FH/VER	12	76.5	102.4	86.38	9.08	24	72.2	101.2	84.34	5.96	-2.04	0.81	0.420
FH/OPT	12	72.3	103.4	89.14	8.55	24	74.4	107.1	88.22	9.08	-0.92	0.29	0.770
FH/CVT	12	77.8	106.6	92.17	7.98	24	81.7	111.0	94.24	8.76	2.07	-0.69	0.490

TABLE 28

HEAD POSTURE IN DEGREES - ANOMALY SAMPLE BEFORE TREATMENT

Value	male n = 31		female n = 41										
	Number	Min	Max	Mean	S.D.	Number	Min	Max	Mean	S.D.	Diff.	t	p
NSL/VER	31	79.4	109.2	97.36	6.43	41	79.8	106.6	96.18	6.09	-1.18	0.80	0.433
NL/VER	31	78.0	100.5	89.63	6.11	41	64.0	97.0	86.44	6.74	-3.19	2.07	0.044
NSL/OPT	31	80.6	114.3	101.72	8.45	41	83.2	119.0	101.18	9.30	-0.54	0.25	0.804
NSL/CVT	31	83.1	118.8	104.22	7.61	41	87.3	123.6	106.42	8.98	2.20	-1.10	0.280
NL/OPT	31	80.3	108.7	94.26	7.49	41	72.8	104.9	91.69	8.81	-2.57	1.30	0.194
NL/CVT	31	85.3	113.2	96.77	6.84	41	77.8	109.5	96.92	8.65	0.15	-0.08	0.941
OPT/HOR	31	72.2	102.8	85.65	7.74	41	71.8	98.6	84.99	6.60	-0.66	0.38	0.703
CVT/HOR	31	72.2	99.0	83.15	6.80	41	65.9	91.7	79.77	6.19	-3.38	2.19	0.034
FH/VER	31	72.0	104.1	87.27	6.54	41	70.0	98.0	84.38	6.24	-2.89	1.19	0.064
FH/OPT	31	73.2	101.2	91.63	7.72	41	70.3	104.3	89.38	8.74	-2.25	1.13	0.263
FH/CVT	31	75.7	105.0	94.12	6.91	41	74.4	109.2	94.61	8.20	0.49	-0.27	0.792

TABLE 29

HEAD POSTURE IN DEGREES - ANOMALY SAMPLE AFTER TREATMENT

Value	Number	male n =31				Number	Min	Max	Mean	S.D.	Min	Max	Mean	S.D.	female n =41		p
															Diff.	t	
NSL/VER	31		83.2	104.5	95.45	5.81	41	85.6	109.2	96.81	5.70	1.36	-0.99	0.325			
NL/VER	31		75.5	98.0	86.97	5.52	41	71.0	96.5	86.79	5.82	-0.18	0.13	0.898			
NSL/OPT	31		80.6	115.6	101.24	8.86	41	88.5	124.4	102.91	8.70	1.67	-0.80	0.426			
NSL/CVT	31		84.8	115.1	103.41	8.02	41	92.6	128.0	108.03	8.12	4.62	-2.40	0.019			
NL/OPT	31		77.6	105.1	93.01	7.51	41	77.8	111.1	93.15	8.26	0.14	-0.07	0.941			
NL/CVT	31		82.2	106.8	95.17	6.77	41	85.9	114.7	98.26	7.82	3.09	-1.76	0.083			
OPT/HOR	31		69.5	106.0	84.21	7.71	41	68.8	98.1	83.90	7.62	-0.31	0.17	0.864			
CVT/HOR	31		69.0	100.3	82.04	6.65	41	65.1	89.9	78.78	6.37	-3.26	2.11	0.038			
FH/VER	31		72.8	96.4	85.59	5.88	41	73.0	94.7	85.01	5.43	-0.58	0.43	0.669			
FH/OPT	31		72.3	104.0	91.39	8.13	41	74.2	107.1	91.11	7.96	-0.28	0.15	0.882			
FH/CVT	31		76.5	103.5	93.55	7.37	41	79.8	110.5	96.25	7.29	2.70	-1.55	0.126			

TABLE 30

COMPARISON OF HEAD POSTURE IN DEGREES BETWEEN CONTROL AND ANOMALY SAMPLES

Variable	Number	Control Sample			Number	Min	Anomaly Before Treatment Sample			t	p	
		Max	Mean	S.D.			Max	Mean	S.D.			Diff.
NSL/VER	36	82.6	110.0	95.34	72	79.4	109.2	96.69	6.22	1.35	-1.02	0.308
NL/VER	36	72.0	104.0	87.68	72	64.0	100.5	87.81	6.63	0.13	-0.09	0.925
NSL/OPT	36	80.8	115.9	98.85	72	80.6	119.0	101.41	8.89	2.56	-1.41	0.161
NSL/CVT	36	86.3	123.5	103.86	72	83.1	123.6	105.47	8.43	1.61	-0.92	0.358
NL/OPT	36	70.0	111.1	91.50	72	72.8	108.7	92.80	8.32	1.30	-0.74	0.462
NL/CVT	36	75.4	118.6	96.50	72	77.8	113.2	96.85	7.87	0.35	-0.21	0.835
OPT/HOR	36	68.5	102.9	86.49	72	71.8	102.8	85.28	7.07	-1.21	0.80	0.428
CVT/HOR	36	64.7	95.4	81.47	72	65.9	99.0	81.23	6.63	-0.24	0.17	0.864
FH/VER	36	72.2	102.4	85.02	72	70.0	104.1	85.63	6.49	0.61	-0.45	0.657
FH/OPT	36	72.3	107.1	88.53	72	70.3	104.3	90.35	8.34	1.83	-1.05	0.295
FH/CVT	36	77.8	111.0	93.55	72	74.4	109.2	94.39	7.63	0.84	-0.53	0.599

D) Airway Dimensions

When the data on airway dimensions was examined to check on the acceptability of combining the mean values for males and females in the two groups of subjects (Tables 31,32,33) it was found that in the anomaly sample before and after treatment, the mean value for pm-ad₃ was significantly larger in males ($p < 0.05$).

In the control group, the mean value of tu-ad₃ was significantly greater in females ($p = 0.012$). Caution must therefore be exercised in pooling male and female values relating to the airway dimensions pm-ad₃ and tu-ad₃.

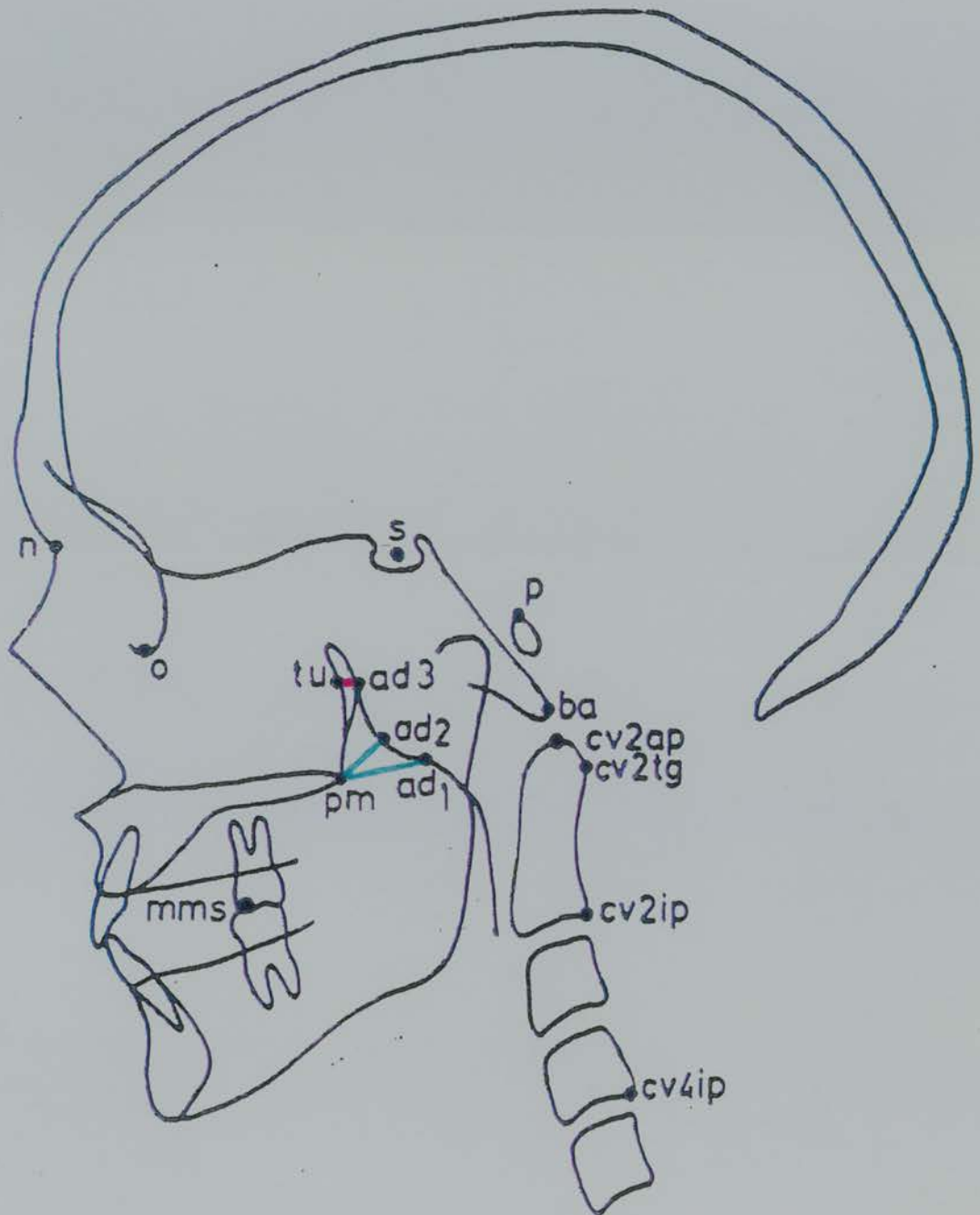
The comparison of the control and anomaly samples before treatment are shown in Table 34.

The mean variables pm-ad₁ and pm-ad₂ were larger in the control group, the differences being highly significant ($p < 0.001$). The actual mean values of pm-ad₁ in the control and anomaly groups were 22.03mm and 18.12mm respectively. For pm-ad₂ the means were 17.29mm and 13.98mm respectively (see Figure 36).

Data for males and females were not combined when the variables pm-ad₃ and tu-ad₃ were considered. Comparison of the control group and the anomaly group for each sex (Tables 35 36) showed that pm-ad₃ was significantly smaller ($p < 0.05$) in the male control sample. The tu-ad₃ differences were highly significant in both males and females ($p < 0.001$), the male control sample mean being 4.03mm against the male anomaly sample before treatment mean value of 6.90mm, a difference of 2.87mm; the female control sample mean value was 4.99mm against the female anomaly before treatment mean value of 6.93mm, a difference of 1.94mm.

There exists a difference in form between the control and anomaly before treatment samples with regard to airway dimensions. The anomaly sample before treatment is narrower in the dimensions $pm-ad_1$ and $pm-ad_2$, but wider at the top of the airway chamber as measured by $tu-ad_3$.

AIRWAY DIMENSION RESULTS



SIGNIFICANT DIFFERENCES

CONTROL SAMPLE/ANOMALY SAMPLE BEFORE TREATMENT

Fig. 36

TABLE 31

AIRWAY DIMENSIONS IN MM - CONTROL SAMPLE

Value	male n = 12			female n = 24			Diff.	t	p
	Number	Min	Max	Mean	S.D.	Number	Mean	S.D.	
pm-ad ₁	12	13.2	26.4	20.06	4.27	24	23.01	6.27	0.154
pm-ad ₂	12	7.6	21.9	16.23	4.66	24	17.82	4.92	0.363
pm-ad ₃	12	14.3	29.1	22.68	4.22	24	22.92	3.44	0.861
tu-ad ₃	12	2.9	5.0	4.03	0.66	24	4.99	1.16	0.012

TABLE 33

AIRWAY DIMENSIONS IN MM - ANOMALY SAMPLE AFTER TREATMENT

Value	male n = 31					female n = 41					t	p	
	Number	Min	Max	Mean	S.D.	Number	Min	Max	Mean	S.D.			Diff.
pm-ad ₁	31	8.0	28.3	19.91	5.39	41	7.5	28.9	19.42	5.58	-0.49	0.37	0.714
pm-ad ₂	31	6.5	22.1	14.84	3.49	41	6.8	21.0	14.67	3.78	-0.17	0.19	0.846
pm-ad ₃	31	21.1	31.0	24.96	2.46	41	12.9	29.1	23.16	3.37	-1.80	2.50	0.015
tu-d ₃	31	5.0	13.0	7.24	1.69	41	3.6	12.9	7.03	1.74	-0.21	0.52	0.605

TABLE 34

COMPARISON OF AIRWAY DIMENSIONS IN MM BETWEEN CONTROL AND ANOMALY SUBJECTS BEFORE TREATMENT

Variable	Control Number	Control Min	Control Max	Control Mean	Control S.D.	Number	Min	Max	Anomaly Before Treatment Max	Anomaly Before Treatment Mean	Anomaly Before Treatment S.D.	Diff.	t	p
pm-ad ₁	36	10.30	35.30	22.03	5.794	72	4.6	29.2	29.2	18.12	5.359	-3.91	3.47	0.001
pm-ad ₂	36	7.60	29.10	17.29	4.825	72	4.6	22.0	22.0	13.98	4.078	-3.31	3.74	0.001
pm-ad ₃	36	14.30	30.50	22.84	3.657	72	15.3	32.3	32.3	23.75	3.083	0.91	-1.35	0.179
tu-ad ₃	36	2.50	7.90	4.51	0.911	72	3.2	12.3	12.3	6.92	1.88	2.40	-6.60	0.001

TABLE 35

COMPARISON OF AIRWAY DIMENSIONS IN MM BETWEEN
MALE CONTROL SUBJECTS AND MALE ANOMALY SUBJECTS BEFORE TREATMENT

Variable	Number	Control Sample		Mean	S.D.	Number	Min	Max	Anomaly Sample Before Treatment		t	p
		Min	Max						Mean	S.D.	Diff.	
pm-ad ₃	12	14.3	29.1	27.68	4.22	31	20.1	32.3	24.74	2.33	-2.06	-2.04
tu-ad ₃	12	2.9	5.0	4.03	0.66	31	3.2	9.6	6.90	1.59	2.87	-6.00
												0.054
												0.001

E) Craniofacial Morphology - Linear Dimensions

Comparisons between male and female mean values of linear hard tissue variables in samples (see Tables 37,38,39) show several significant differences. The variable sp-pm (maxillary length) was significantly larger in males than in females in both samples. The overbite in the control sample was significantly deeper in the male group than the female ($p=0.02$).

In all other linear hard tissue variables, with the exception of n-sp, oj and ob, statistically significant differences were found in the anomaly sample before and after treatment, the differences for n-s, n-gn, s-ba, s-pm, s-tgo, sp-gn being significant at the $p<0.001$ level. The male values were consistently larger, reflecting the general difference in size between males and females in the chosen age band of 10 to 15 years inclusive.

It was concluded that it would be preferable to consider the data for each sex separately.

The comparison of the linear craniofacial variables of the control and the anomaly sample before treatment with RME (Tables 40,41,42) showed a number of statistically significant differences (see Figure 37).

In those values which relate to the height of the anterior facial complex, the upper facial height n-sp, lower facial height sp-gn and total anterior facial height n-gn, all showed significantly different mean values in the male subjects only (see Table 41), being greater in the anomaly sample before treatment than in the control: n-sp had a mean difference of 2.88mm ($p=0.039$); sp-gn a mean difference of 5.97mm ($p<0.001$); and n-gn a mean difference of 9.68mm ($p<0.001$).

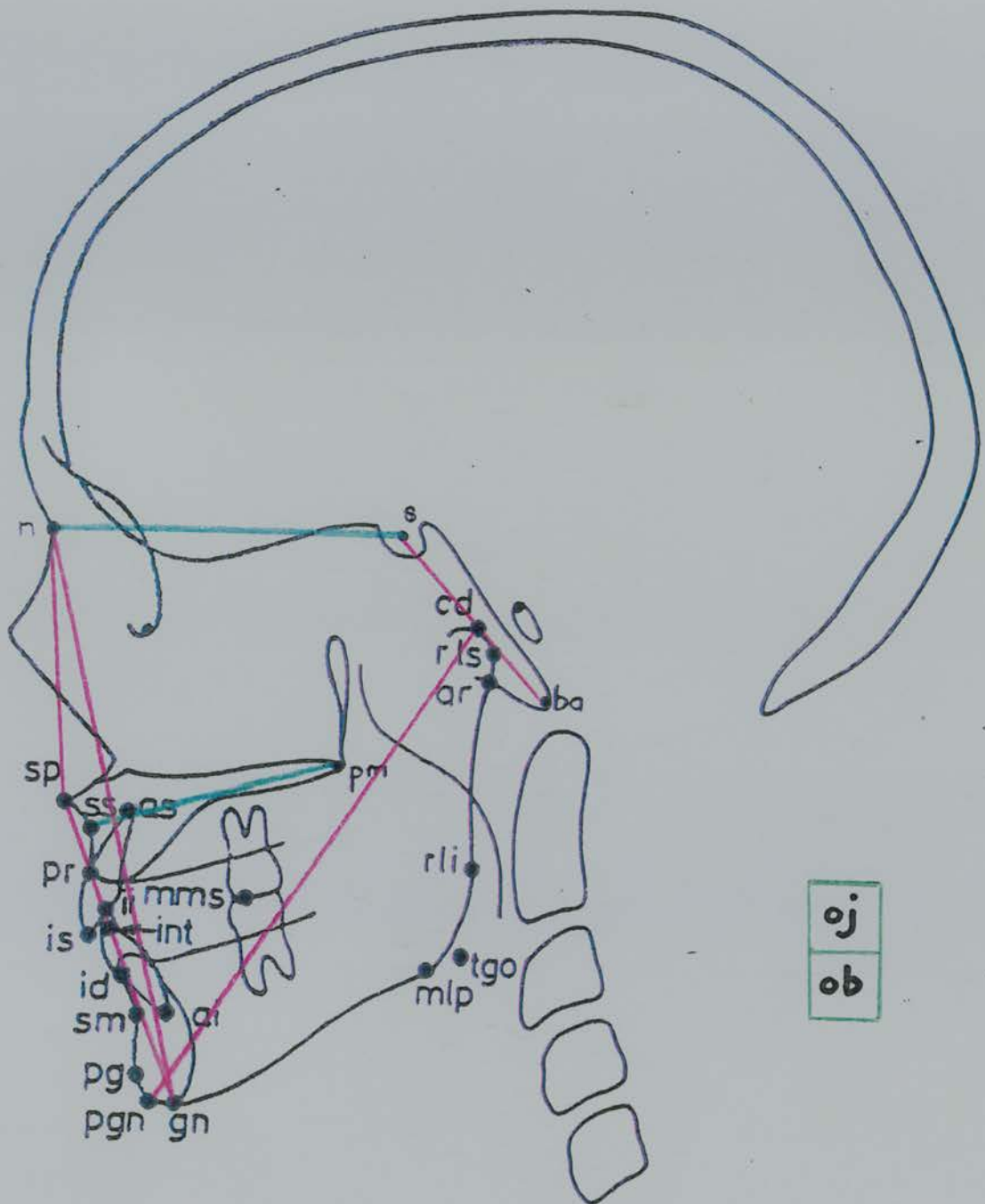
Also, only in the male subjects (see Table 41), the clivus length s-ba was significantly ($p=0.022$) larger in the anomaly sample before treatment by a mean difference of 2.93mm. For both males and females, the value of the variable

pgn-cd, a measurement of mandibular length, was also significantly greater ($p<0.001$) in the anomaly sample, there being an overall mean difference of 7.27mm.

However in two of the linear variables that measure facial depth, n-s and ss-pm, the values for the total anomaly sample were significantly less than those for the control sample, but when the sexes were considered separately the differences were statistically significant in the female subjects only (see Table 42). In females the variable n-s had a mean difference of 2.83mm ($p=0.003$) and ss-pm a mean difference of 2.09mm ($p=0.006$).

The overjet and overbite linear values were significantly different between the two samples in both males and females (see Tables 41,42). The overjet in the combined control sample was larger by a mean difference of 3.82mm ($p<0.001$) and the overbite deeper in the combined control sample by a mean difference of 2.50mm ($p<0.001$) (see Table 40).

CEPHALOMETRIC LINEAR RESULTS



SIGNIFICANT DIFFERENCES

CONTROL SAMPLE/ANOMALY SAMPLE BEFORE TREATMENT

Fig. 37

TABLE 37

CRANIOFACIAL MORPHOLOGY - LINEAR VALUES IN MM - CONTROL SAMPLE

Value	male n = 12			female n = 24			Diff.	t	p
	Number	Min	Max	Mean	Max	S.D.			
n-s	12	69.9	81.3	73.95	81.9	3.21	0.02	-0.02	0.993
n-sp	12	47.1	56.2	52.66	59.5	3.13	0.64	-0.54	0.594
n-gn	12	110.8	127.7	118.56	133.1	5.18	0.54	-0.23	0.822
s-ba	12	38.3	50.3	45.93	50.4	3.22	-0.80	0.58	0.563
s-pm	12	43.3	57.1	49.34	54.0	3.87	-1.14	0.84	0.414
sp-gn	12	61.8	75.3	68.35	81.3	4.32	-0.36	0.19	0.854
sp-pm	12	50.9	61.9	56.56	61.5	3.54	-2.62	2.22	0.032
ss-pm	12	47.5	55.8	51.90	55.5	2.51	-1.37	1.55	0.131
pgn-cd	12	106.9	126.2	115.67	125.8	4.82	-1.26	0.59	0.564
oj	12	3.8	9.9	6.41	11.4	1.87	-0.13	0.17	0.863
ob	12	-7.3	-3.5	-4.83	0.4	0.94	-1.31	-2.46	0.024

TABLE 38

CRANIOFACIAL MORPHOLOGY - LINEAR VALUES IN MM - ANOMALY SAMPLE BEFORE TREATMENT

Value	Number	male n = 31			S.D.	Number	Min	Max	female n = 41			Diff.	t	p
		Min	Max	Mean					Mean	S.D.				
n-s	31	64.9	80.3	73.63	3.56	41	62.0	78.0	71.14	3.51	-2.49	2.96	0.004	
n-sp	31	48.3	62.2	55.54	4.23	41	46.7	62.7	54.44	3.55	-1.10	1.19	0.240	
n-gn	31	115.5	146.4	128.24	8.16	41	108.0	136.7	121.74	7.30	-6.50	3.55	0.001	
s-ba	31	41.8	55.9	48.85	3.72	41	40.3	50.9	45.87	2.75	-2.98	3.91	0.001	
s-pm	31	42.1	55.8	49.85	3.49	41	39.9	54.4	46.96	3.08	-2.89	3.73	0.001	
sp-gn	31	67.9	87.3	74.32	4.87	41	59.1	84.4	68.56	6.12	-5.76	4.31	0.001	
sp-pm	31	48.8	62.7	55.61	3.54	41	47.0	59.8	43.29	3.15	-12.32	2.92	0.005	
ss-pm	31	40.5	59.0	50.07	3.59	41	42.8	55.5	48.44	3.05	-1.63	2.08	0.041	
pgn-cd	31	112.9	135.6	124.54	7.42	41	107.4	133.9	120.25	6.30	-4.29	2.65	0.010	
oj	31	-6.2	12.2	2.95	4.24	41	-3.4	9.7	2.18	3.94	-0.77	0.80	0.430	
ob	31	-6.4	3.7	-1.79	2.36	41	-5.9	2.5	-1.21	2.44	0.58	-1.03	0.310	

TABLE 39

CRANIOFACIAL MORPHOLOGY - LINEAR VALUES IN MM - ANOMALY SAMPLE AFTER TREATMENT

Value	Number	male n = 31			S.D.	Number	Min	Max	female n = 41			Diff.	t	p
		Min	Max	Mean					Mean	S.D.	Mean			
n-s	31	65.7	80.5	73.92	3.64	41	61.7	78.3	71.05	3.52		-2.87	3.37	0.001
n-sp	31	45.9	65.2	56.61	4.56	41	46.5	63.8	55.17	3.64		-1.44	1.49	0.142
n-gn	31	117.7	146.6	130.80	8.01	41	108.3	137.8	124.16	6.94		-6.64	3.76	0.001
s-ba	31	41.2	55.5	48.95	3.67	41	41.8	50.8	45.97	2.65		-2.98	4.01	0.001
s-pm	31	44.2	55.6	50.35	3.37	41	41.5	54.8	47.64	2.82		-2.71	3.72	0.001
sp-gn	31	67.9	89.6	76.13	4.52	41	58.9	83.1	70.49	5.76		-5.64	4.50	0.001
sp-pm	31	47.4	62.3	55.37	3.98	41	46.3	61.4	53.08	3.06		-2.29	2.77	0.007
ss-pm	31	37.5	59.0	49.86	3.95	41	41.5	55.0	48.31	3.09		-0.55	1.87	0.066
pgn-cd	31	114.1	138.0	124.87	7.19	41	108.1	134.9	120.69	6.19		-4.18	2.64	0.010
oj	31	-5.0	11.5	3.76	3.66	41	-5.2	9.8	2.47	3.94		-1.29	1.43	0.158
ob	31	-6.6	3.6	-0.22	2.79	41	-5.2	3.5	0.08	2.49		0.14	-0.47	0.640

TABLE 41

COMPARISON OF CRANIOFACIAL LINEAR VARIABLES IN MM BETWEEN
MALE CONTROL SUBJECTS AND MALE ANOMALY SUBJECTS BEFORE TREATMENT

Variable	male control sample				male sample before treatment				t	p			
	Number	Min	Max	Mean	S.D.	Number	Min	Max			Mean	S.D.	Diff.
n-s	12	69.9	81.3	73.95	3.21	31	64.9	80.3	73.63	3.56	-0.32	0.27	0.787
n-sp	12	47.1	56.2	52.66	3.13	31	48.3	62.2	55.54	4.23	2.88	-2.13	0.039
n-gn	12	110.8	127.7	118.56	5.18	31	115.5	146.4	128.24	8.16	9.68	-3.80	0.001
s-ba	12	38.3	50.3	45.93	3.22	31	41.8	55.9	48.85	3.72	2.93	-2.39	0.022
s-pm	12	43.3	57.1	49.34	3.87	31	42.1	55.8	49.85	3.49	0.51	-0.42	0.679
sp-gn	12	61.8	75.3	68.35	4.32	31	67.9	87.3	74.32	4.87	5.97	-3.72	0.001
sp-pm	12	50.9	61.9	56.56	3.54	31	48.8	62.7	55.61	3.54	-0.95	0.79	0.434
ss-pm	12	47.5	55.8	51.90	2.51	31	40.5	59.0	50.07	3.59	-1.83	1.61	0.114
pgn-cd	12	106.9	126.2	115.67	4.82	31	112.9	135.6	124.54	7.42	8.87	-3.83	0.001
oj	12	3.8	9.9	6.41	1.87	31	- 6.2	12.2	2.95	4.24	-3.46	2.71	0.010
ob	12	- 7.3	- 3.5	- 4.83	0.94	31	- 6.4	3.7	- 1.79	2.36	-3.04	-4.31	0.001

F) Craniofacial Morphology - Angular Dimensions

Comparison of males and females in the groupings showed very few statistically significant differences between the sets of mean variables (Tables 43,44,45)

In view of the small number of these significant differences, it was concluded that it would be acceptable to combine data from the sexes when undertaking future comparisons, exercising some caution with the angle n-s-ba, which was significantly greater in females in the anomaly group.

Analysis of the comparison between the control sample and the anomaly sample before treatment revealed a high number of significant differences (see Table 46). The angle pm-s-ba was significantly ($p=0.003$) smaller in the anomaly sample, the mean difference being 3.47° - this correlates with previous results in this study, showing the maxillary position to be an important cause of the decrease in nasopharyngeal space in the anomaly sample before treatment. The values s-n-sp and s-n-ss also showed significantly smaller values in the anomaly sample ($p<0.05$ and $p<0.001$) the mean differences being 1.60° and 2.91° respectively; this shows that the maxillary complex was less prominent in the anomaly sample when compared to the control group. Further, the angular relationship between the maxilla and mandible, ss-n-sm and ss-n-pg, both showed highly significant differences ($p<0.001$ and $p=0.017$ respectively), the anomaly sample having a smaller value with means of 3.78° and 1.01° respectively. The maxillary complex was thus set further back in relation to the mandible in the anomaly sample.

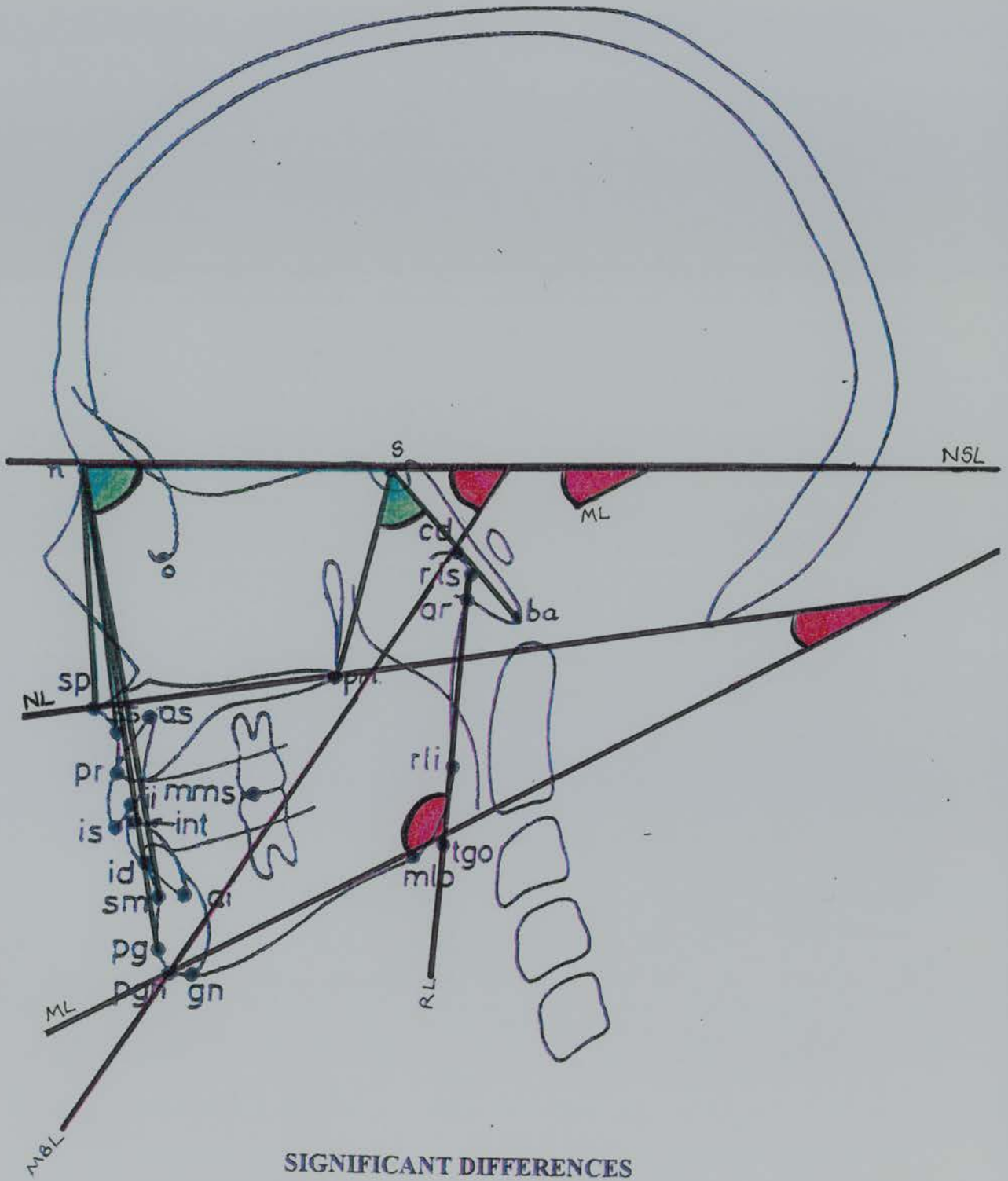
The antero-posterior position of the mandible in relation to s-n measured by the angles s-n-sm and s-n-pg, showed no significant differences.

The angle NSL/NL showed no significant difference between the two samples, indicating that the maxilla, although somewhat distal in the anomaly sample, was not tipped when measured against the control. However, the angle NSL/ML was highly significantly different ($p=0.002$); the mean value in the

anomaly group was larger by an average of 3.70° , as was the NL/ML angle ($p=0.027$), the mean value in the anomaly sample being larger by 2.44° over the control group mean.

Finally, the angles NSL/MBL and ML/RL were both significantly greater ($p<0.006$ and $p<0.000$) in the anomaly sample when compared to the control sample; the average differences were 2.78° and 4.27° respectively (see Figure 38.)

CEPHALOMETRIC ANGULAR RESULTS



CONTROL SAMPLE/ANOMALY SAMPLE BEFORE TREATMENT

Fig. 38

TABLE 43

CRANIOFACIAL MORPHOLOGY - ANGULAR VALUES IN DEGREES - CONTROL SAMPLE

Value	male n = 12				female n = 24				Diff.	t	p
	Number	Min	Max	Mean	S.D.	Number	Min	Max			
n-s-ba	12	116.2	134.8	128.08	5.44	24	121.9	145.5	2.80	-1.43	0.164
pm-s-ba	12	47.8	65.0	56.91	6.10	24	50.9	70.0	-2.43	-1.14	0.263
s-n-sp	12	79.8	95.0	88.01	4.73	24	76.0	96.8	-3.57	2.04	0.051
s-n-ss	12	75.3	87.7	82.78	4.47	24	72.1	91.0	-2.15	1.43	0.163
s-n-sm	12	72.4	83.5	78.45	3.17	24	69.5	85.4	-1.90	1.44	0.164
s-n-pg	12	73.8	84.2	79.60	3.03	24	68.5	84.4	-2.46	1.87	0.071
ss-n-sm	12	0.5	9.6	4.32	2.79	24	-0.9	11.1	-0.26	0.29	0.774
ss-n-pg	12	0.4	8.1	3.30	2.54	24	0.4	9.8	0.56	-0.69	0.493

TABLE 43 (cont)

CRANIOFACIAL MORPHOLOGY - ANGULAR VALUES IN DEGREES - CONTROL SAMPLE

Value	male n = 12			female n = 24			Diff.	t	p
	Number	Min	Max	Mean	Max	Mean			
NSL/NL	12	1.0	13.0	6.19	15.5	7.94	1.75	-1.26	0.224
NSL/ML	12	26.0	40.3	31.68	46.0	35.00	-3.32	-1.94	0.064
NL/ML	12	16.2	35.6	25.49	35.7	25.05	-0.44	-0.81	0.423
NSL/MBL	12	52.6	65.4	57.70	67.1	60.02	2.32	-1.79	0.092
ML/RL	12	112.0	136.4	125.45	138.1	125.87	0.42	-0.18	0.861

TABLE 44 (cont)

CRANIOFACIAL MORPHOLOGY - ANGULAR VALUES IN DEGREES
ANOMALY SAMPLE BEFORE TREATMENT

Value	male n = 31					female n = 41					t	p	
	Number	Min	Max	Mean	S.D.	Number	Min	Max	Mean	S.D.			Diff.
NSL/NL	31	-2.6	13.4	7.44	3.74	41	1.4	15.9	9.49	3.76	2.05	-2.29	0.032
NSL/ML	31	19.9	46.6	37.40	6.05	41	27.1	49.9	37.73	5.86	-0.33	-0.24	0.814
NL/ML	31	18.3	40.6	29.97	5.24	41	18.1	41.1	28.24	5.29	-1.73	1.38	0.173
NSL/MBL	31	49.2	70.1	61.65	4.59	41	51.5	71.6	61.88	4.95	0.23	-0.21	0.844
ML/RL	31	118.9	139.7	130.64	5.83	41	121.6	146.7	130.95	5.33	0.31	-0.23	0.824

TABLE 45

CRANIOFACIAL MORPHOLOGY - ANGULAR VALUES IN DEGREES
ANOMALY SAMPLE AFTER TREATMENT

Value	male n = 31				female n = 41				Diff.	t	p
	Number	Min	Max	Mean	S.D.	Number	Min	Max			
n-s-ba	31	116.4	137.5	127.57	4.45	41	114.8	145.1	3.11	-2.29	0.025
pm-s-ba	31	45.7	65.4	53.62	4.53	41	44.3	68.3	2.35	-2.05	0.044
s-n-sp	31	75.4	94.5	84.75	4.42	41	75.6	93.7	-1.20	1.22	0.227
s-n-ss	31	70.0	88.0	79.07	4.21	41	72.0	88.5	-0.59	0.63	0.531
s-n-sm	31	71.3	88.6	77.28	4.51	41	67.7	88.8	0.30	-0.28	0.782
s-n-pg	31	71.9	89.9	78.19	4.27	41	69.1	89.5	-0.06	0.06	0.954
ss-n-sm	31	-5.6	7.8	1.78	3.43	41	-5.7	6.1	-0.88	1.13	0.262
ss-n-pg	31	0.3	7.8	3.38	1.92	41	0.1	7.4	-0.53	1.85	0.216

TABLE 45 (cont)

CRANIOFACIAL MORPHOLOGY - ANGULAR VALUES IN DEGREES
ANOMALY SAMPLE AFTER TREATMENT

Value	male n = 31			female n = 41					Diff.	t	p
	Number	Min	Max	Mean	S.D.	Number	Min	Max			
NSL/NL	31	-0.3	16.9	8.24	3.87	41	0.8	15.5	1.52	-1.68	0.097
NSL/ML	31	22.8	47.6	38.55	6.55	41	27.5	52.8	0.53	-0.36	0.722
NL/ML	31	17.4	41.4	30.33	5.79	41	16.2	42.1	-1.01	0.76	0.452
NSL/MBL	31	52.0	70.7	62.77	4.88	41	51.6	73.7	0.02	-0.01	0.990
ML/RL	31	119.3	138.9	130.64	6.03	41	123.3	146.8	0.68	-0.51	0.613

TABLE 46

A COMPARISON OF CRANTIOFACIAL ANGULAR VALUES IN DEGREES BETWEEN THE
CONTROL SUBJECTS AND THE ANOMALY SUBJECTS BEFORE TREATMENT

Variable	Control Sample Number	Min	Max	Mean	S.D.	Number	Min	Max	Anomaly Sample Before Treatment Mean	S.D.	Diff.	t	p
n-s-ba	36	116.2	145.5	129.95	5.594	72	116.0	145.3	130.01	5.828	0.06	-0.06	0.956
pm-s-ba	36	47.8	70.0	58.62	6.409	72	44.5	71.1	55.15	5.013	-3.47	3.09	0.003
s-n-sp	36	76.0	96.8	85.63	5.164	72	74.9	94.4	83.79	4.213	-1.60	1.98	0.050
s-n-ss	36	72.1	91.0	81.34	4.318	72	71.1	88.8	78.43	3.940	-2.91	3.51	0.001
s-n-sm	36	69.5	85.4	77.18	3.789	72	67.9	91.4	78.07	4.622	0.89	-1.00	0.318
s-n-pg	36	68.5	84.4	77.96	3.840	72	69.7	92.8	78.88	4.530	0.92	-1.04	0.299
ss-n-sm	36	-0.9	11.1	4.14	2.482	72	-6.1	6.3	0.36	3.016	-3.78	6.50	0.001
ss-n-pg	36	0.4	9.8	3.68	2.301	72	0.1	7.0	2.67	1.857	-1.01	2.44	0.017
NSL/NL	36	-0.1	15.5	7.36	3.953	72	-2.6	15.9	8.61	3.863	1.25	-1.57	0.119
NSL/ML	36	26.0	46.0	33.89	5.023	72	19.9	49.9	37.59	5.903	3.70	-3.22	0.002
NL/ML	36	16.2	35.7	26.54	5.438	72	18.1	41.1	28.98	5.303	2.44	-2.24	0.027
NSL/MBL	36	52.6	67.1	59.24	3.846	72	49.2	71.6	61.79	4.764	2.55	-2.78	0.006
ML/RL	36	112.0	138.1	125.73	6.455	72	118.9	146.7	130.82	5.512	5.09	-1.27	0.001

G) Correlations

The relationship between head posture, craniocervical angulation and craniofacial morphology was the subject of a hypothesis suggested by Solow and Kreiborg (1977) (see Figure 1). It was suggested that subjects exhibiting increased nasal airway resistance extended the head in relation to the cervical column in order to maintain their vital airway adequacy. This head extension led in turn to stretching of the facial soft tissues, resulting in differential forces on the facial skeleton with consequent effect on craniofacial growth. The net result was the "adenoidal facies".

An analysis of the correlation between specific variables relating to anterior facial height and head posture reveals a comprehensive pattern of associations in the anomaly sample before treatment (see Table 48.) The head posture variables NSL/CVT and NSL/OPT showed positive associations with upper facial height n-sp, lower facial height sp-gn and NL/ML, the angular measurement of lower facial height. Hence a large facial height was seen with a large craniofacial angulation.

Negative associations were seen in the anomaly sample before treatment and in the control sample between the head posture variables and the measurements of airway adequacy pm-ad₁ and pm-ad₂. Hence the larger the head posture variables, the smaller the airway dimension value.

It would appear, therefore, that the present study goes a considerable way to supporting the Solow and Krieborg (1977) control system hypothesis.

TABLE 47

CORRELATIONS

CONTROL SAMPLE N = 36

	Facial morphology			Head posture		Airway dimensions				Nasal resistance		Width			
	n-sp	sp-gn	NL/ML	NSL/CVT	NSL/OPT	pm-ad ₁	pm-ad ₂	pm-ad ₃	tu-ad ₃	PNR (I)	PNR (E)	CNR/CML	Maxilla	Nasal	EMR/EML
n-sp
sp-gn	.	.	0.59**	-0.36*	-0.35*	.	.	.	0.39*
NL/ML	.	0.59**
NSL/CVT	0.94**
NSL/OPT	.	.	.	0.94**
pm-ad ₁	0.80*
pm-ad ₂	-0.39*	0.80**
pm-ad ₃	0.56**	.	0.56**
tu-ad ₃
PNR (I)	-0.36*
PNR (E)	-0.35*	0.98**
CNR/CNL	0.98**
EMR/EML	0.39*	0.33*	.	.	0.33*

* p < .05
** p < .01

TABLE 48

CORRELATIONS

ANOMALY SAMPLE BEFORE TREATMENT N = 72

	Facial morphology			Head posture			Airway dimensions			Nasal resistance		Width	
	n-sp	sp-gn	NL/ML	NSLCVT	NSL/OPT	pm-ad ₁	pm-ad ₂	pm-ad ₃	tu-ad ₃	PNR(I)	PNR(E)	Maxilla	Nasal
n-sp
sp-gn	0.36**	.	.	0.35**	0.39**
NL/ML	.	0.62**	.	0.25*	0.32**	.	.	0.29*
NSL/CVT	.	0.62**	.	0.43**	0.41**
NSL/OPT	0.39**	0.32**	0.41**	.	.	-0.31**	-0.37**
pm-ad ₁	.	.	.	-0.31**	.	.	0.86**	0.36**	0.24**
pm-ad ₂	.	.	.	-0.36**	-0.37**	0.86**	0.52**	0.52**	0.32**
pm-ad ₃	.	0.29*	.	.	.	0.36**	0.52**	0.40**
tu-ad ₃	0.24*	0.32**
PNR(I)	0.99**	.	.	.
PNR(E)	0.99**	.	.	.
CNR/CNL	0.49**	.
EMR/EML	.	.	.	-0.27*	-0.29*	.	.	0.40**	0.49**

* p < 0.5

** p < 0.1

5.2 CHANGES IN THE ANOMALY SAMPLE WITH TREATMENT

A) Nasal Airway Resistance

Comparisons of the mean rhinomanometric variables between males and females in the anomaly sample after treatment (see Tables 19 and 20) showed only four statistically significant differences ($p < 0.05$) out of a total of eighteen separate comparisons. It was therefore concluded that it would be reasonable to combine the data for the sexes in this group.

Total upper airway resistance

The changes in the rhinomanometric values of the anomaly sample are shown in Table 4950. As far as the total upper airway values are concerned, the anomaly sample before treatment showed a mean value of 468.76 for inspiration, against the anomaly sample after treatment value of 395.44, a reduction of 73.32. This is a significant difference at the $p < 0.001$ level, as is the total expiratory mean value of 459.97 for the anomaly sample before treatment compared with 375.67 for the sample after treatment, a reduction of 84.30. (see Fig. 39)

The change in the total laminar (K_1) value for inspiration was from 196.84 for the anomaly sample before treatment to 130.29 after treatment, a reduction of 66.55 ($p < 0.001$) and for the expiratory (K_1) laminar flow value from 186.53 before treatment to 123.16 after treatment, a reduction of 63.37 ($p < 0.001$). The change in the mean turbulent (K_2) flow value for inspiration was from 714.85 before treatment to 482.47 after treatment - a reduction of 232.38 ($p < 0.001$); for expiration the mean was 710.56 before treatment compared with a mean of 490.81 after treatment; a reduction of 219.75 ($p < 0.001$). (Fig. 40)

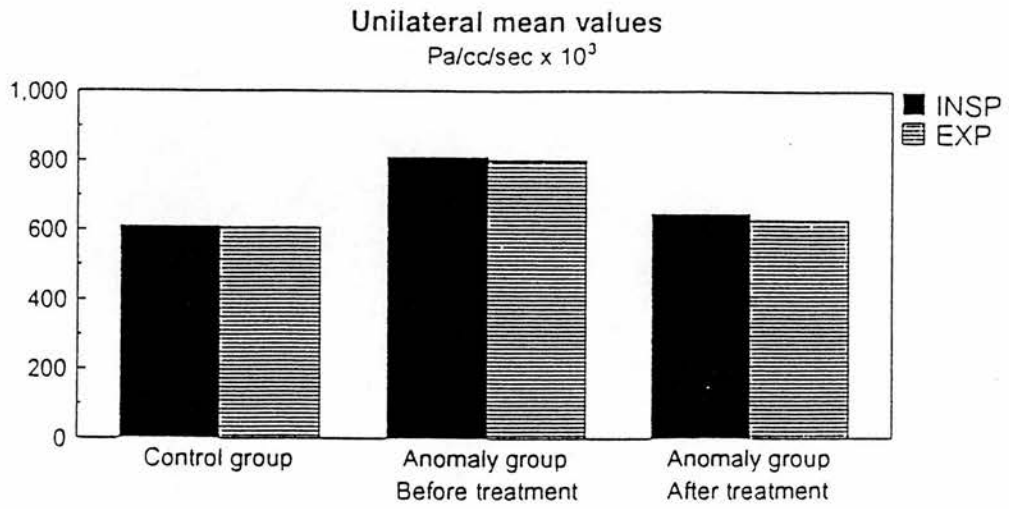
Unilateral nasal airway resistance

As can be seen from Table 49 the mean differences in NAR in the anomaly sample before and after treatment are all highly significant at the $p < 0.001$ level, with the mean values after treatment being the lower in each case. The mean values of the resistance before treatment varied from 819.48 pascals/cc/second $\times 10^3$ for NR R EXP to 827.98 pascals/cc/second $\times 10^3$ for NR R INS. The mean values for the resistance after treatment varied from 631.68 pascals/cc/second $\times 10^3$ for NA R EXP to 656.69 pascals/cc/second $\times 10^3$ for NA L INS. (see Fig.39)

For laminar flow values (K_1) and turbulent flow values (K_2) (see Table 50) the mean differences were also seen to be highly statistically significant at the $p < 0.001$ level. The laminar flow values before treatment ranged from 239.43 for K_1 L EXP to 267.27 for K_1 R INS. The mean laminar flow values after treatment varied from 165.66 for K_1 L EXP to 188.05 for K_1 R INS.

The mean turbulent flow values before treatment ranged from 1233.87 for K_2 R EXP to 1328.92 for K_2 L INS. After treatment the range was from 888.91 for K_2 R EXP to 1012.80 for K_2 L INS. (see Fig.40)

Nasal Airway Resistance



Nasal Airway Resistance

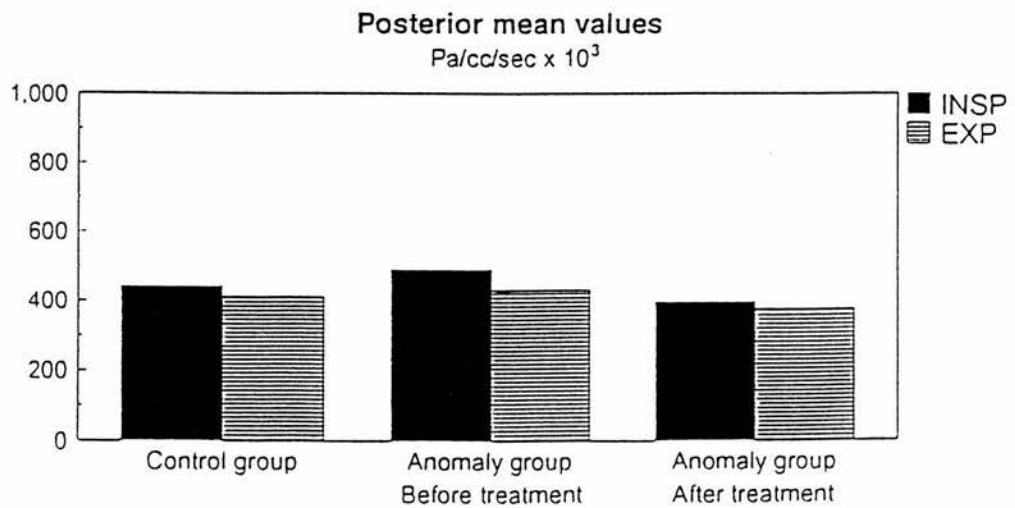
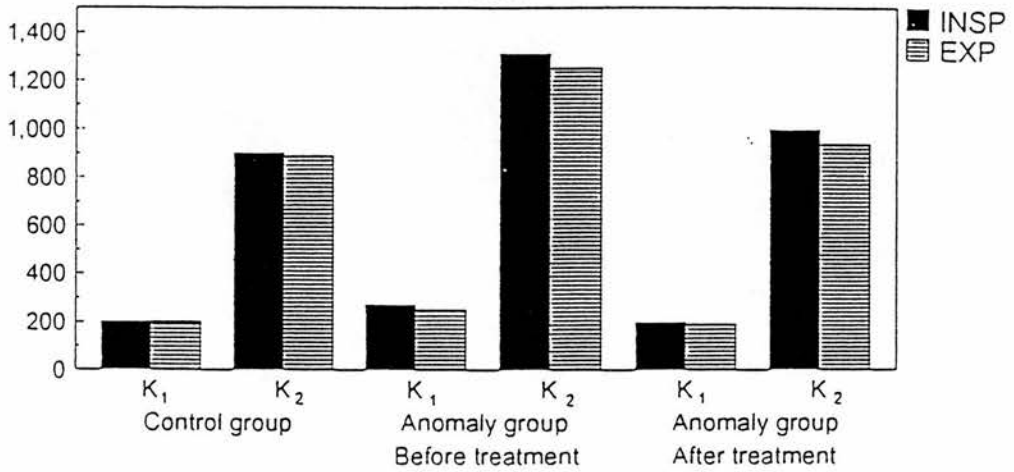


Fig. 39

Nasal Airway Resistance

Unilateral Laminar & Turbulent mean values

$\text{Pa/cc/sec} \times 10^3$



Nasal Airway Resistance

Posterior Laminar & Turbulent mean values

$\text{Pa/cc/sec} \times 10^3$

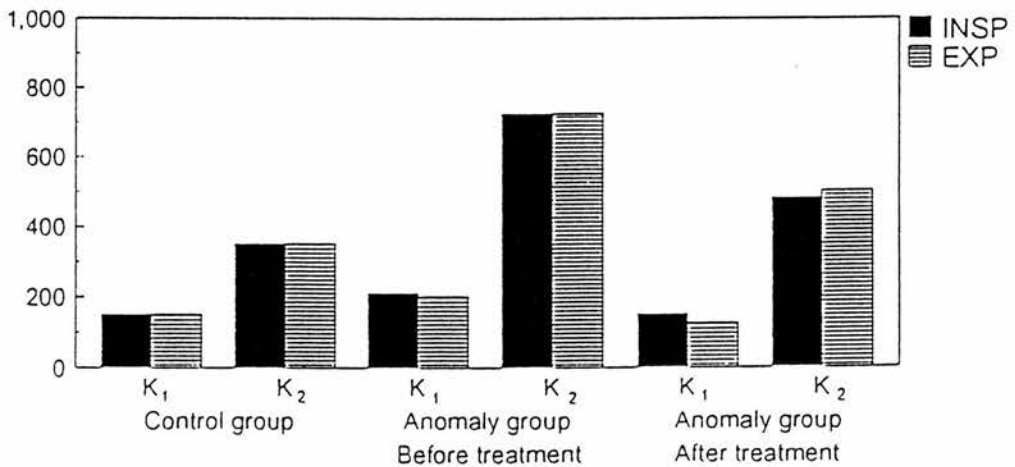


Fig. 40

TABLE 49

NASAL AIRWAY RESISTANCE: TREATMENT CHANGES IN ANOMALY SAMPLE
N A R (pascal/cc/sec $\times 10^3$) at 150 pascals

Variable	Number*	Anomaly Before Min	Max	Mean	S.D.	Number*	Min	Max	Anomaly After Mean	S.D.	Diff.	t	p
<u>ANT L</u>													
INSP	57	460	1746	827.89	230.66	57	411	996	656.69	140.68	-171.20	7.18	0.001
EXP	57	425	1668	825.47	227.54	57	410	957	637.11	139.07	-188.36	7.71	0.001
<u>ANT R</u>													
INSP	55	447	1565	827.98	227.77	55	356	1037	644.17	144.99	-183.88	6.78	0.001
EXP	55	438	1514	819.48	229.08	55	335	1069	631.68	154.76	-187.80	6.80	0.001
<u>POST</u>													
INSP	58	302	971	468.76	147.27	58	249	883	395.44	108.79	-73.32	4.68	0.001
EXP	58	293	900	459.97	140.29	58	222	851	375.67	106.03	-84.30	4.50	0.001

* The numbers vary because certain subjects were unable to complete all the tests

TABLE 50

NASAL AIRWAY RESISTANCE: TREATMENT CHANGES IN ANOMALY SAMPLE
Laminar and Turbulent flow coefficients K1/K2 (pascal/cc/sec x 10³) at 150 pascals

Variable	Before Treatment			After Treatment			Mean	S.D.	Diff.	t	p
	Number*	Min	Max	Min	Max	Number*					
<u>ANT L</u>											
INSP K ₁	59	120	593			59	176.82	44.77	-80.48	7.56	0.001
EXP K ₁	59	110	410	93	246	59	165.66	41.27	-73.77	7.93	0.001
<u>ANT R</u>											
INSP K ₁	55	110	876			55	188.05	57.89	-79.22	4.17	0.001
EXP K ₁	55	108	848	105	368	55	180.47	55.13	-76.33	3.97	0.001
<u>POST</u>											
INSP K ₁	58	102	484			58	130.29	44.71	-66.55	5.83	0.001
EXP K ₁	58	97	478	56	220	58	123.16	40.52	-63.37	5.44	0.001
<u>ANT L</u>											
INSP K ₂	59	452	1933			59	1012.80	311.98	-316.12	7.41	0.001
EXP K ₂	59	447	1934	512	1773	59	981.09	300.13	-295.50	7.27	0.001
<u>ANT R</u>											
INSP K ₂	55	374	1907			55	941.68	362.25	-355.93	6.55	0.001
EXP K ₂	55	397	1887	343	1752	55	888.91	351.19	-344.96	6.44	0.001
<u>POST</u>											
INSP K ₂	58	177	1571			58	482.47	193.52	-232.38	5.59	0.001
EXP K ₂	58	176	1512	116	1040	58	490.81	194.61	-219.75	5.09	0.001

* The numbers vary because certain subjects were unable to complete all the tests

B) Transverse Craniofacial Dimensions

These results will be presented in two ways; first, with the sexes considered separately and, second, a comparison of the changes to the combined sex anomaly sample.

(i) Sexes considered separately

When the variables relating to the two sexes were compared in the anomaly sample after treatment (see Table 25), a number of changes showed significant differences between the mean values for males and females. It was thought that this may indicate a different response by males and females to the process of rapid maxillary expansion, so comparisons were made (see Tables 52 and 53) between male subjects before and after treatment and, separately, female subjects before and after treatment.

On analysis of the results, however, the male and female groups both largely followed the same trend as indicated by the combined results table (Table 51), so the existence of a fundamental difference between males and females in the reaction to rapid palatal expansion could not be shown.

(ii) Sexes combined

When the comparison was made of the mean postero-anterior cephalometric variables in the anomaly sample before treatment and after treatment (see Table 51), several statistically significant differences were found (see Figure 41).

The total mean width in mm of the anterior nasal base CNR-CNL was significantly larger ($p < 0.001$) in the sample after treatment, by about 10.5%. When this measurement is broken down into right side (CNR-CNM) and left side (CNM-CNL) both are found to be significant at the $p < 0.001$ level, the right side showing an increase of 7.8%, the left 8.9%. (The apparent discrepancy of the upper left and right sides increasing by some 8%, whilst the total width increases by 10%, reflects the problem of accurately measuring the right and left components.)

The maxillary box width EMR-EML was significantly increased ($p < 0.001$) after treatment in both males and females, a 3.5% difference, whereas the distances between the buccal surfaces of the maxillary first molars UMR-UML increased by 14.5%, significant at the $p < 0.001$ level in both males and females.

The total width in mm between the buccal surfaces of the lower first molars, LMR-LML, also increased significantly overall after treatment ($p < 0.002$) (Table 51).

The distance in m.m. between the mesial surfaces of the two upper central incisors, MIR-MIL before and after treatment, showed a highly significant difference ($p < 0.001$), as did the breakdown of the two subsections MIR-C₂ and C₂-MIL ($p < 0.001$).

Finally, the nasal proportion NAS DIM showed an increase of 3.75% after treatment with RME ; significant at the $p < 0.001$ level.

TABLE 51

CHANGES IN TRANSVERSE CEPHALOMETRIC DIMENSIONS IN MM WITH TREATMENT OF ANOMALY SAMPLE

Variable	Anomaly Sample Before Treatment					Anomaly Sample After Treatment					t	p	
	Number	Min	Max	Mean	S.D.	Number	Min	Max	Mean	S.D.			Diff.
CNR-CNM	72	12.80	19.90	15.85	1.700	72	14.60	21.50	17.08	1.495	1.23	-8.41	0.001
CNM-CNL	72	11.90	20.00	15.46	1.670	72	11.60	20.80	16.84	1.650	1.38	-9.71	0.001
CNR-CNL	72	20.20	35.00	28.00	2.948	72	24.50	37.50	30.96	2.851	2.96	-17.93	0.001
EMR-EML	72	51.50	68.80	60.99	4.271	72	54.40	76.60	63.14	4.041	2.15	-8.14	0.001
UMR-UML	72	44.20	66.20	53.86	4.579	72	50.20	72.50	61.63	4.578	7.77	-18.35	0.001
LMR-LML	72	44.40	70.70	58.45	4.340	72	44.50	70.80	58.80	4.303	0.35	-3.18	0.002
MIR-MIL	72	0.00	5.30	0.38	0.991	72	2.30	8.20	5.13	1.536	4.75	-23.07	0.001
MIR-C2	72	0.00	2.40	0.19	0.418	72	1.10	3.90	2.46	0.796	2.27	-22.12	0.001
C2-MIL	72	0.00	3.40	0.21	0.577	72	1.10	4.30	2.67	0.787	2.46	-22.70	0.001
NAS-DIM	72	55.50	79.50	66.99	5.258	72	56.00	82.50	69.49	5.732	2.50	6.36	0.001
MAX-DIM	72	68.40	89.50	78.31	5.373	72	68.00	90.30	78.61	5.238	0.30	0.79	0.433

TABLE 52

CHANGES IN TRANSVERSE CEPHALOMETRIC DIMENSIONS IN MM WITH TREATMENT MALE ANOMALY SAMPLE

Variable	Number	Before Treatment				S.D.	Number	Min	Max	Mean	S.D.	After Treatment Diff.	t	p
		Min	Max	Mean	S.D.									
CNR-CNM	31	12.80	19.5	16.23	1.76	31	14.7	21.5	17.41	1.58	+0.82	-4.59	0.001	
CNM-CNL	31	11.90	20.0	15.92	1.88	31	14.4	20.8	17.22	1.48	+1.25	-5.37	0.001	
CNR-CNL	31	23.10	35.0	28.75	2.98	31	24.5	37.3	31.51	2.72	+2.76	-10.21	0.001	
EMR-EML	31	53.60	68.8	62.65	4.19	31	56.9	76.6	64.60	4.51	+1.95	-4.58	0.001	
UMR-UML	31	44.20	66.2	54.93	4.99	31	51.7	72.5	62.93	5.44	+8.00	-12.57	0.001	
LMR-LML	31	48.90	70.7	59.70	4.30	31	49.9	70.8	60.05	4.29	+0.35	-2.18	0.038	
MIR-MIL	31	0.00	5.3	0.58	1.38	31	2.7	7.7	5.54	1.49	+4.96	-15.18	0.001	
MIR-C2	31	0.00	2.4	0.27	0.58	31	1.1	3.9	2.69	0.76	+2.42	-15.12	0.001	
C2-MIL	31	0.00	3.4	0.34	0.81	31	1.5	4.2	2.85	0.77	+2.51	-14.51	0.001	
NAS-DIM	31	58.30	79.0	67.67	4.59	31	58.3	82.5	70.18	5.11	+2.51	3.84	0.001	
MAX-DIM	31	68.40	86.8	78.13	4.67	31	68.0	90.3	78.28	5.06	+0.15	0.24	0.815	

TABLE 53

CHANGES IN TRANSVERSE CEPHALOMETRIC DIMENSIONS IN MM WITH TREATMENT FEMALE ANOMALY SAMPLE

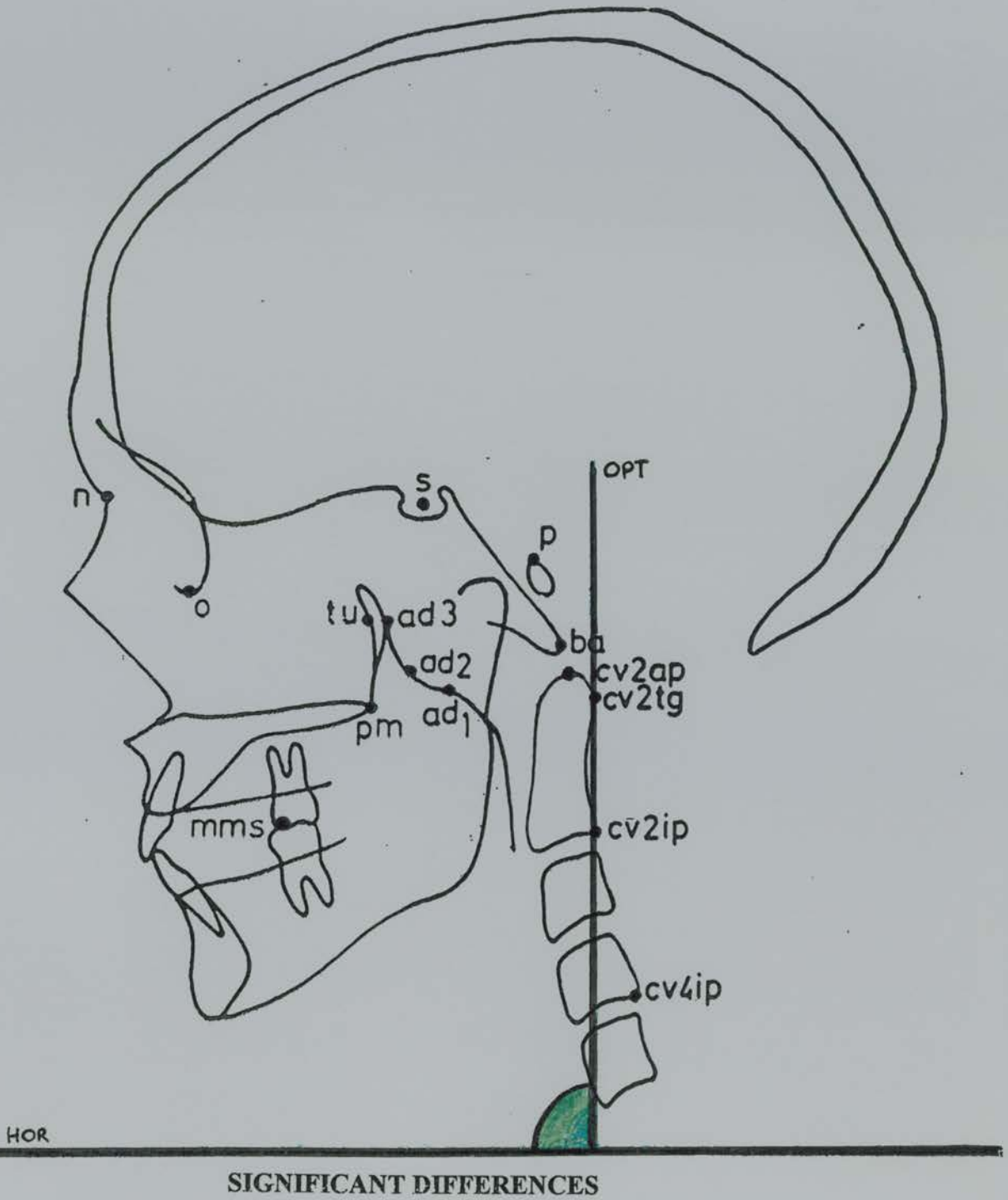
Variable	Number	Before Treatment			S.D.	Number	Min	Max	Mean	After Treatment			t	p
		Min	Max	Mean						S.D.	Diff.	Mean		
CNR/CNM	41	12.90	19.90	15.48	1.65	41	14.60	20.20	16.75	1.41	+1.27	16.75	-7.43	0.001
CNM-CNL	41	12.50	18.10	14.94	1.46	41	11.60	20.20	16.45	1.82	+1.51	16.45	-8.29	0.001
CNR-CNL	41	20.20	33.00	27.44	2.83	41	25.00	37.50	30.55	2.91	+3.11	30.55	-15.15	0.001
EMR-EML	41	51.50	67.80	59.74	3.93	41	54.40	71.00	62.03	3.29	+2.29	62.03	-6.85	0.001
UMR-UML	41	44.20	60.20	53.05	4.12	41	50.20	71.00	62.03	3.29	+7.59	62.03	-13.27	0.001
LMR-LML	41	44.40	67.24	57.51	4.17	41	44.50	68.10	57.86	4.11	+0.35	57.86	-2.31	0.026
MIR-MIL	41	0.00	2.30	0.22	0.51	41	2.30	8.20	4.82	1.51	+4.60	4.82	-17.33	0.001
MIR-C2	41	0.00	1.20	0.13	0.23	41	1.10	3.90	2.29	0.78	+2.16	2.29	-16.29	0.001
C2-MIL	41	0.00	1.40	0.12	0.29	41	1.10	4.30	2.54	0.78	+2.42	2.54	-17.32	0.001
NAS-DIM	41	55.50	79.50	66.48	5.71	41	56.00	82.00	68.97	0.89	+2.49	68.97	5.10	0.001
MAX-DIM	41	69.10	89.50	78.45	5.90	41	69.00	80.00	78.87	5.42	+0.42	78.87	0.88	0.382

C) Head Posture

The changes between the values before and after treatment (see Table 54) were not statistically significant. On examining the three variables previously described, NSL/VER shows a reduction between the means of 0.47 of one degree, NSL/OPT an increase of 0.78 of one degree between the means and NSL/CVT an increase of 0.57 of one degree between the means before and after treatment. All of these differences are negligible.

Only the variable OPT/HOR, however, the cervical inclination, showed a significantly smaller value after treatment with RME ($p=0.032$), the value decreasing from 85.28° to 84.03° , a difference of 1.25° . This difference represents a slight forward tipping of the cervical inclination after treatment (see Figure 42).

HEAD POSTURE RESULTS



ANOMALY SAMPLE BEFORE/AFTER TREATMENT

Fig. 42

TABLE 54

CHANGES IN HEAD POSTURE IN DEGREES WITH TREATMENT

Variable	Number	Min	Anomaly Before			S.D.	Number	Min	Max	Mean	S.D.	Anomaly After Diff.	t	p
NSL/VER	72	79.4	Max	Mean	Max	6.22	72	83.2	109.2	96.22	5.75	-0.47	0.82	0.416
NL/VER	72	64.0	109.2	96.69	100.5	6.63	72	71.0	98.0	86.86	5.65	-0.95	1.67	0.099
NSL/OPT	72	80.6	119.0	101.41	8.89	72	80.6	124.4	102.19	8.75	0.78	-1.19	0.237	
NSL/CVT	72	83.1	123.6	105.47	8.43	72	84.8	128.0	106.04	8.35	0.57	-0.89	0.377	
NL/OPT	72	72.8	108.7	92.80	8.32	72	77.6	111.1	93.09	7.89	0.29	-0.44	0.661	
NL/CVT	72	77.8	113.2	96.85	7.87	72	82.2	114.7	96.93	7.49	0.08	-0.12	0.904	
OPT/HOR	72	71.8	102.8	85.28	7.07	72	68.8	106.0	84.03	7.61	-1.25	2.19	0.032	
CVT/HOR	72	65.9	99.0	81.23	6.63	72	65.1	100.3	80.18	6.65	-1.05	1.97	0.052	
FH/VER	72	70.0	104.1	85.63	6.49	72	72.8	96.4	85.26	5.59	-0.37	0.62	0.536	
FH/OPT	72	70.3	104.3	90.35	8.34	72	72.3	107.1	91.23	7.98	0.88	-1.32	0.192	
FH/CVT	72	74.4	109.2	94.39	7.63	72	76.5	110.5	95.08	7.39	0.69	-1.03	0.304	

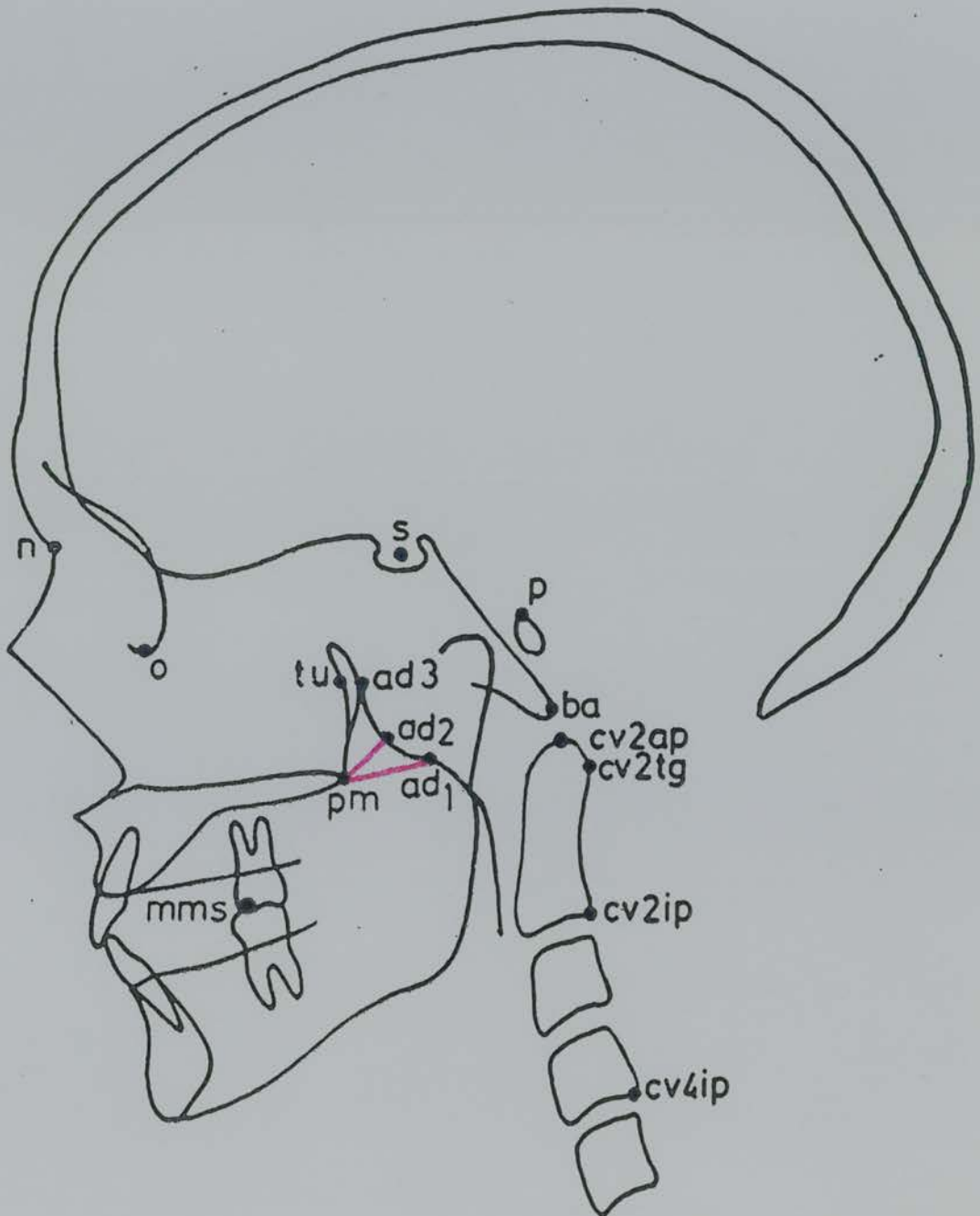
D) Airway Dimensions

Two variables, pm-ad₁ and pm-ad₂, showed highly significant changes with treatment ($p < 0.001$) (see Table 55 and Figure 43).

The mean of pm-ad₁ was increased by 1.51mm, representing an increase in dimension of 8.33%. The value of pm-ad₂ was increased by 0.76mm, representing a 5.43% change.

The other two variables pm-ad₃ and tu-ad₃ showed no significant changes, whether the values were gender pooled or whether males and females were considered separately (Tables 56,57).

AIRWAY DIMENSION RESULTS



SIGNIFICANT DIFFERENCES

ANOMALY SAMPLE BEFORE/AFTER TREATMENT

Fig. 43

TABLE 56

CHANGES IN AIRWAY DIMENSIONS IN MM OF THE MALE ANOMALY SUBJECTS WITH TREATMENT

Variable	Anomaly Sample Before Treatment				Number	Min	Anomaly Sample After Treatment				t	p
	Number	Min	Max	Mean			Max	Mean	S.D.	Diff.		
pm-ad ₃	31	20.1	32.3	24.74	31	21.1	31.0	24.96	2.46	0.22	-0.62	0.543
tu-ad ₃	31	3.2	9.6	6.90	31	5.0	13.0	7.24	1.69	0.34	-1.01	0.320

TABLE 57

CHANGES IN AIRWAY DIMENSIONS IN MM OF THE FEMALE ANOMALY SUBJECTS WITH TREATMENT

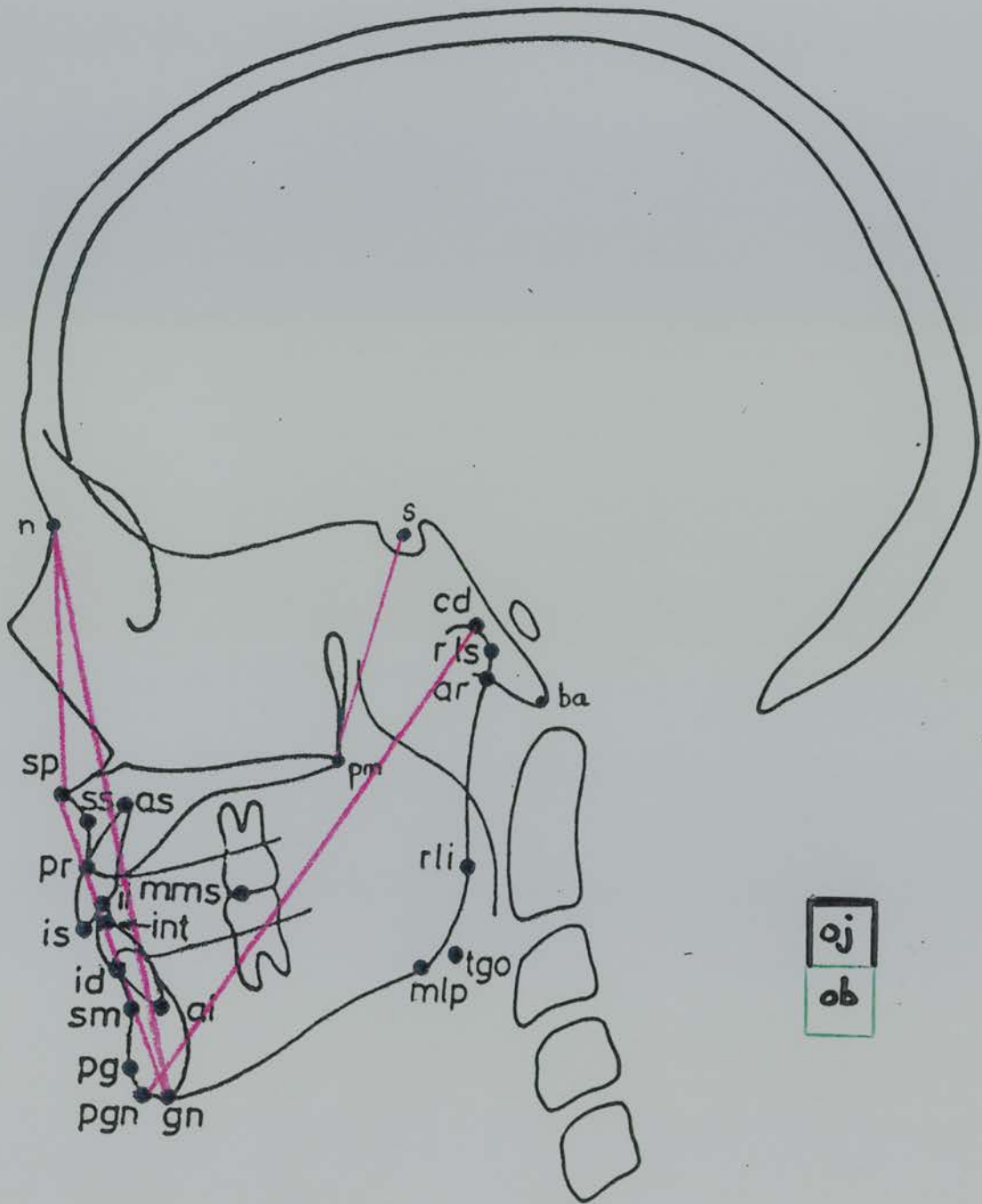
Variable	Anomaly Sample Before Treatment				Anomaly Sample After Treatment				t	p			
	Number	Min	Max	S.D.	Number	Min	Max	Mean			S.D.	Diff.	
pm-ad ₃	41	15.3	29.1	22.99	3.39	41	12.9	29.1	23.16	3.37	0.17	-0.50	0.617
tu-ad ₃	41	3.7	12.3	6.93	2.09	41	3.6	12.9	7.03	1.74	0.10	-0.41	0.687

E) Craniofacial Morphology - Linear Dimensions

After treatment with RME, all the anterior linear facial measurements increased significantly in both males and females (see Tables 58,59,60). n-sp showed a combined mean difference of 0.88mm ($p < 0.001$); sp-gn showed a combined mean difference of 1.88mm ($p < 0.001$); and n-gn a combined mean difference of 2.48mm ($p < 0.001$). Of particular interest, the variable s-pm between sella and the distal point on the maxilla also showed a significantly larger mean value in the anomaly sample after treatment for both male and female groupings, the combined mean difference being 0.61mm ($p < 0.001$). This value has relevance in the movement of the maxilla as a result of treatment, and in the relative sizes of the nasopharyngeal space before and after treatment.

The variables n-s and ss-pm that measure facial depth showed no significant change in the total anomaly sample and in females (see Tables 58,60); the only significant difference was that of n-s ($p < 0.05$) in the male group (see Table 59). The overbite reduced significantly in both sexes ($p < 0.001$) after treatment, by an overall mean difference of 1.41mm (see Figure 44).

CEPHALOMETRIC LINEAR RESULTS



SIGNIFICANT DIFFERENCES

ANOMALY SAMPLE BEFORE/AFTER TREATMENT

Fig. 44

TABLE 58

CHANGES IN CRANIOFACIAL LINEAR VARIABLES IN MM BETWEEN
THE ANOMALY SUBJECTS BEFORE AND AFTER TREATMENT

Variable	Anomaly Sample Before Treatment				Number	Min	Max	Mean	S.D.	Number	Min	Max	Anomaly Sample After Treatment			p
													Mean	S.D.	Diff.	
n-s	72	62.0	80.3	72.21	3.724	72	61.7	80.5	3.826	0.08	-0.72	0.477				
n-sp	72	46.7	62.7	54.91	3.870	72	45.9	65.2	4.095	0.88	-5.00	0.001				
n-gn	72	108.0	146.4	124.54	8.287	72	108.3	146.6	8.076	2.48	-8.89	0.001				
s-ba	72	40.3	55.9	47.16	3.510	72	41.2	55.5	3.447	0.09	-0.79	0.435				
s-pm	72	39.9	55.8	48.20	3.546	72	41.5	55.6	3.333	0.61	-3.90	0.001				
sp-gn	72	59.1	87.3	71.04	6.277	72	58.9	89.6	5.933	1.88	-7.84	0.001				
sp-pm	72	47.0	62.7	54.29	3.497	72	46.3	62.3	3.647	-0.22	1.19	0.239				
ss-pm	72	40.5	59.0	49.14	3.868	72	37.5	59.0	3.542	-0.83	1.10	0.276				
pgn-cd	72	107.4	135.6	122.10	7.084	72	108.1	138.0	6.912	0.39	-2.56	0.013				
oj	72	-7.4	12.2	2.51	4.058	72	-5.2	11.5	3.849	0.52	-1.65	0.104				
ob	72	-6.4	3.7	-1.46	2.404	72	-6.6	3.6	2.611	1.41	-5.64	0.001				

TABLE 59

CHANGES IN CRANIOFACIAL LINEAR VARIABLES IN MM BETWEEN THE
MALE ANOMALY SUBJECTS BEFORE AND AFTER TREATMENT

Variable	male sample before treatment					male sample after treatment					t	p	
	Number	Min	Max	Mean	S.D.	Number	Min	Max	Mean	S.D.			Diff.
n-s	31	64.9	80.3	73.63	3.56	31	65.7	80.5	73.92	3.64	0.29	-2.13	0.041
n-sp	31	48.3	62.2	55.54	4.23	31	45.9	65.2	56.61	4.56	1.07	-3.71	0.001
n-gn	31	115.5	146.4	128.24	8.16	31	117.7	146.6	130.80	8.01	2.56	-5.49	0.000
s-ba	31	41.8	55.9	48.85	3.72	31	41.2	55.5	48.95	3.67	0.10	-0.55	0.583
s-pm	31	42.1	55.8	49.85	3.49	31	44.2	55.6	50.35	3.37	0.50	-2.68	0.012
sp-gn	31	67.9	87.3	74.32	4.87	31	67.9	89.6	76.13	4.52	1.81	-5.46	0.001
sp-pm	31	48.8	62.7	55.61	3.54	31	47.4	62.3	55.37	3.98	-0.24	0.75	0.460
ss-pm	31	40.5	59.0	50.07	3.59	31	37.5	59.0	49.86	3.95	-0.21	0.93	0.362
pgn-cd	31	112.9	135.6	124.54	7.42	31	114.1	138.0	124.87	7.19	0.33	-1.00	0.326
oj	31	- 6.2	12.2	2.95	4.24	31	- 5.0	11.5	3.76	3.66	0.81	-1.52	0.140
ob	31	- 6.4	3.7	- 1.79	2.36	31	- 6.6	3.6	-0.22	2.79	-1.57	-3.81	0.001

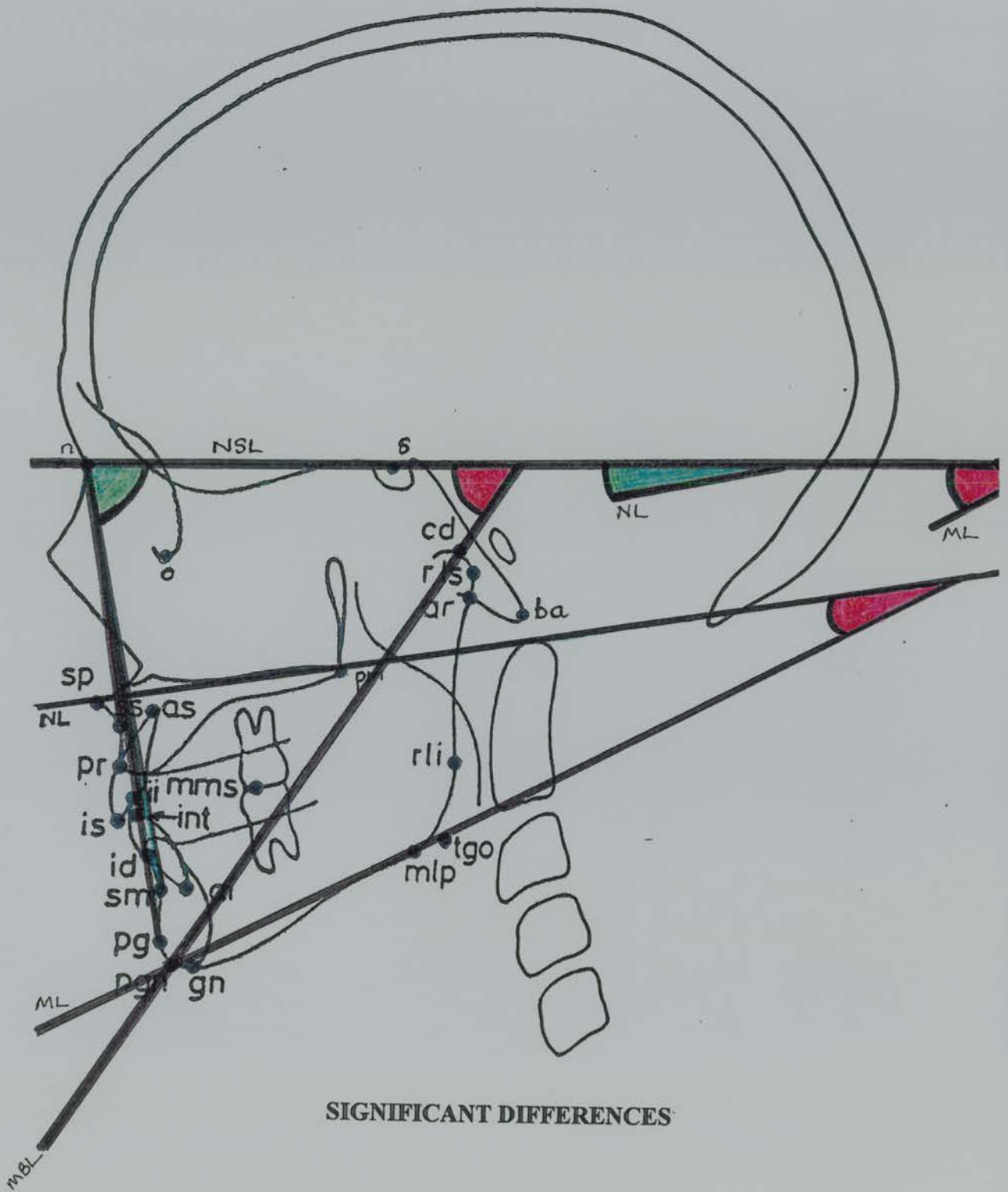
F) Craniofacial Morphology - Angular Dimensions

The analysis of the significant differences within the anomaly sample before and after RME (see Table 61) reveal one surprise - the fact that the angle pm-s-ba showed no statistically significant difference between the two samples; it was expected that, given the increase in dimensions pm-ad₁ and pm-ad₂ after RME, the point pm would have moved forward as a result of the expansion. However, point pm moved only downwards to increase the dimensions of the nasopharyngeal space (see relevance of value s-pm noted previously).

There was some slight distal tipping downwards of NL; in fact the angle NSL/NL did show a statistically significant reduction between the two means, a small mean difference of 0.5° ($p=0.026$).

The mandibular protrusion relative to the cranial base (s-n-sm and s-n-pg) was significantly reduced ($p<0.001$) with treatment, perhaps due to the auto-rotation of the mandible. The sagittal jaw relation angles ss-n-sm and ss-n-pg were found to be significantly greater after treatment ($p<0.001$ and $p=0.016$). The angle of the mandible to the cranial base NSL/ML increased significantly ($p<0.001$) after treatment, a mean difference of 1.27° - this is consistent with the slight increase in lower facial height previously noted, and with the repositioning of the mandible. Clinically it was probably due to the posterior propping open of the bite as the change from bilateral crossbite to normal bite was achieved. This may also account for the significant difference between NL/ML ($p=0.003$) and NSL/MBL ($p<0.001$) (see Figure 45).

CEPHALOMETRIC ANGULAR RESULTS



ANOMALY SAMPLE BEFORE/AFTER TREATMENT

Fig. 45

TABLE 61
CHANGES IN CRANIOFACIAL ANGULAR VALUES IN DEGREES IN THE ANOMALY SUBJECT WITH TREATMENT

Variable	Anomaly Sample Before Treatment					Anomaly Sample After Treatment					p		
	Number	Min	Max	Mean	S.D.	Number	Min	Max	Mean	S.D.		Diff.	t
n-s-ba	72	116.0	145.3	130.01	5.828	72	114.8	137.5	129.84	5.887	-0.17	0.07	0.937
pm-s-ba	72	44.5	71.1	55.15	5.013	72	44.3	68.3	54.96	4.911	-0.19	0.85	0.396
s-n-sp	72	74.9	94.4	83.79	4.213	72	75.4	94.5	84.06	4.150	0.27	-1.27	0.208
s-n-ss	72	71.1	88.8	78.43	3.940	72	70.0	88.5	78.74	3.890	0.31	-1.96	0.054
s-n-sm	72	67.9	91.4	78.07	4.622	72	67.7	88.8	77.45	4.486	-0.62	3.45	0.000
s-n-pg	72	69.7	92.8	78.88	4.530	72	69.1	89.9	78.16	4.470	-0.72	3.84	0.001
ss-n-sm	72	-6.1	6.3	0.36	3.016	72	-5.7	7.8	1.28	3.267	0.92	-4.88	0.001
ss-n-pg	72	0.1	7.0	2.67	1.857	72	0.1	7.8	3.08	1.810	0.41	-2.47	0.016
NSL/NL	72	-2.6	15.9	8.61	3.863	72	-0.3	14.9	8.10	3.837	-0.51	-2.27	0.026
NSL/ML	72	19.9	49.9	37.59	5.903	72	22.8	52.8	38.86	6.246	1.27	-5.38	0.001
NL/ML	72	18.1	41.1	28.98	5.303	72	16.2	42.1	29.75	5.585	0.77	-3.13	0.003
NSL/MBL	72	49.2	71.6	61.79	4.764	72	51.6	73.7	62.78	5.003	0.99	-4.50	0.001
ML/RL	72	118.9	146.7	130.82	5.512	72	119.3	146.8	131.03	5.615	0.21	-1.29	0.200

6. DISCUSSION

The present study was concerned with the difference between craniofacial, head posture and nasal airway resistance values shown by an anomaly sample of seventy two children aged 10 and 15 years (inclusive), and a control sample of thirty six children of matching age. The anomaly sample subjects all exhibited a full transverse cusp bilateral crossbite. Further, the study then examined and tested the changes that occurred to the anomaly sample subjects when they were treated using rapid maxillary expansion, and considered the inter-relationship between these changes. None of the subjects from either the anomaly or control sample had undergone any surgery to the nasal, tonsillar or adenoidal regions.

The method errors for all the measurement systems were tested and found to be satisfactory both in terms of the equipment used and the ability of the operator to reproduce measurements. The study followed the recommendations of the Committee Report on Standardisation of Rhinomanometry (Clement 1984) and adopted S.I. units, all recordings for nasal airway resistance (N.A.R.) being shown in Pascals/cc/second $\times 10^3$, the resistance value being calculated at a point on the curve corresponding to a pressure difference of 150 Pascals. Rhinomanometry generally is subject to method errors that are larger than those observed in other measurements. Therefore particular care was taken with this aspect of the study, and has resulted in method error values that compare well with previous studies (Solow and Greve 1980, and Sandham 1988). The accuracy whilst measuring N.A.R. was enhanced by using a modified Scuba mask (Hansen et al 1984) and by utilising visual feedback for the patient, a computerised trace on the screen showing the mean of four respiratory cycles (Solow and Greve 1980). The utilisation of an adapted Scuba mask for the pneumotachograph had additional positive points - notably the acceptance by children of a familiar object and

the fact that it only covered the nose, allowing the oral tubing to measure the total nasal resistance to be slightly adapted to obtain optimum readings. However some children found the mask uncomfortable and claustrophobic and were not able to complete readings on all tests. Others found some difficulty in accepting the oral tube for the posterior measurement - this factor was overcome by modifying the tubing with a sleeve, coupled with the Bio-feedback techniques with visual screen presentation.

The NR6 Rhinomanometer was calibrated before each session and proved to be very reliable and robust.

The Rohrer chip in the computer enabled the N.A.R. to be subdivided into values for laminar (k_1) and turbulent (k_2) components and for these values to be compared between the control sample and anomaly sample, and the anomaly sample before and after treatment with RME. This subdivision into laminar (k_1) and turbulent (k_2) values is particularly meaningful when the differing flow patterns of inspiratory and expiratory air are considered. On inspiration, the nasal valve acts as the main resistor in the nasal airway (Proctor 1977; Haight and Cole 1983; Warren et al 1987) causing turbulence in the airflow. This resistance at the nasal valve is regulated by the circumferential alar muscles, and by the erectile tissue on the adjacent septum and turbinates (Hasegawa et al 1979). The airflow then normally passes through the middle meatus, the air being warmed and humidified on passing over the highly vascularised turbinates. When the turbinates are approximate to the nasal septum, as here in the anomaly sample before treatment, there is restriction of airflow, and the nasal blanket stagnates (see Figure 46A). This leads to chemical changes in the composition of the mucopolysaccharides on the nasal mucosa, leading to secondary bacterial rhinosinusitis. The turbulence engendered on inspiration allows the nose to function correctly,

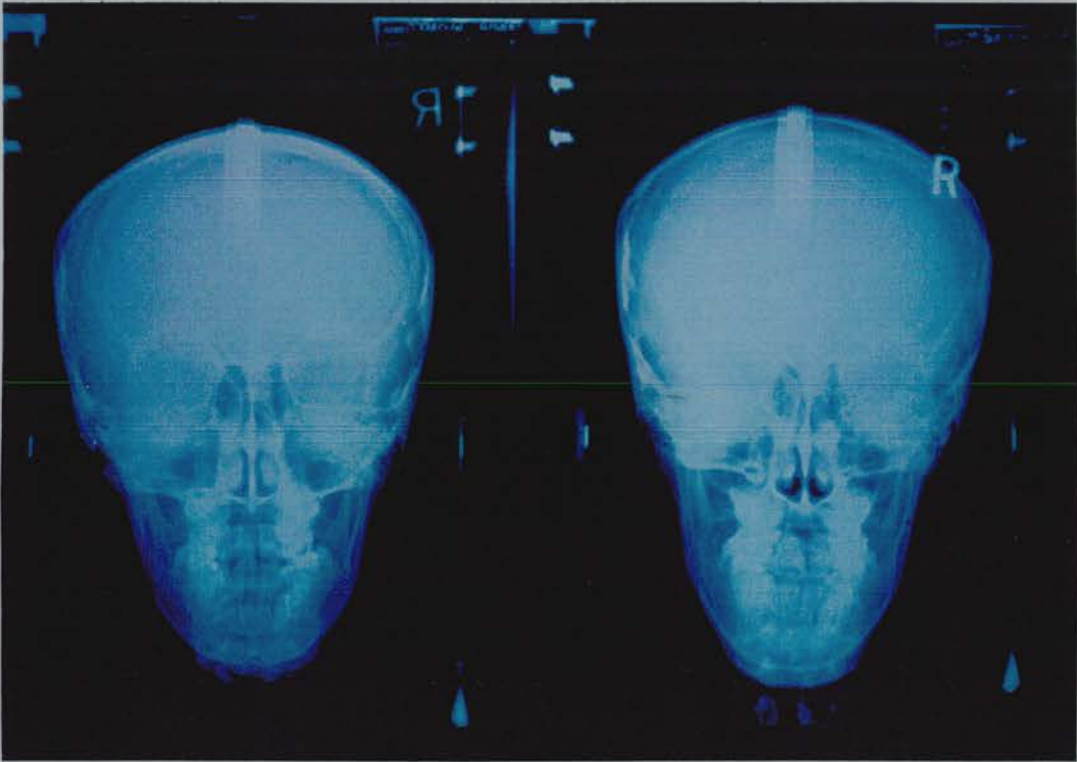


Fig. 46 Effect on intranasal transverse dimensions of treatment with RME

warm, moist filtered air being presented to the delicate alveoli of the lungs, as well as allowing an eddy airflow to pass over the olfactory membrane, and a small flow of air along the floor of the nose (Cole and Haight 1984).

Expired air from the lungs is passed through the nose as a result of a passive deflation of the lungs, and does not require to be warmed, humidified or filtered.

It is important that non-invasive instrumentation is utilised whilst measuring N.A.R., as any invasive technique causes an artificial departure from norm function (Rao and Potdar 1981; Weber et al 1981; Strohl et al 1982; Cole and Haight 1984). In this study, a small diameter pressure recording tube was attached by adhesive tape to the nasal aperture, hence avoiding the necessity of inserting a recording tube into the nares, with consequent distortion of the readings.

Differences between the control sample and the anomaly sample

In the present study, comparison of the N.A.R. of the control sample and anomaly sample before treatment (see Table 21) showed a significantly higher ($p < 0.001$) anterior N.A.R. in the anomaly sample than in the control, a difference of about 200 Pascals/cc/second $\times 10^3$ in both the inspiratory and expiratory modes; the difference in the posterior values was less significant ($p = 0.069$) in the region of 50 Pascals/cc/second $\times 10^3$, as was the laminar flow ($p < 0.005$), these mean values being some 30% higher in the anomaly sample. However, when the values for turbulent flow were appraised, a more significant difference was shown ($p < 0.001$) for both anterior and total values, the mean values of the anomaly sample before treatment being in the region of 50% higher than those in the control sample. There clearly exists, therefore, a significant and demonstrable difference in N.A.R. between the control sample

and the anomaly sample. The readings relating to turbulent flow (k_2) (see Table 22) are in particular significantly higher in the anomaly sample, underlying the importance of accurate measurement of this parameter and the possibility of turbulent flow being a causative factor in the stimulation of mouth breathing as opposed to nasal breathing in a high demand situation.

The airway dimensions pm-ad₁ and pm-ad₂ in the anomaly sample (which had demonstrably higher nasal airway resistance) were found to be significantly smaller ($p < 0.001$), a difference of 21.5% and 20.8% respectively, than in the control sample, even though the linear dimension tu-ad₃ was larger. The position of the point pm, the most posterior point on the maxilla, would appear to be relevant, it being in a more distal position in the anomaly sample in relation to other craniofacial structures when compared to the control sample. This is supported by the fact that the angle pm-s-ba is significantly smaller ($p = 0.003$) in the anomaly sample than in the controls.

If the transverse variables are considered, then a comparison between the control sample and the anomaly sample before treatment shows a number of statistically significant differences at the $p < 0.001$ and $p < 0.005$ levels. The measurement CNR-CNL, which is the transverse width of the anterior nasal floor, has a mean value of 29.64mm in the control sample and 28.00mm in the anomaly sample, a difference significant at the $p < 0.005$ level. The value EMR-EML, the transverse width of the maxillary base, is significantly smaller in the anomaly sample ($p < 0.001$) and the measurement between the upper first molars UMR-UML was also significantly smaller ($p < 0.001$) in the anomaly sample. Finally, the dimensions NASDIM and MAXDIM both showed significance ($p < 0.001$), the anomaly sample being smaller.

Hence the term "adenoidal facies" that was used to describe children with long narrow faces caused, it was thought, primarily by enforced long term mouth breathing, gains support from this study. However, the nasal stenosis causing

the oral breathing pattern here comes not from the adenoidal hypertrophy as described by Linder-Aronson (1970), but by a combination of distal positioning of the point pm (see Table 34) and a significantly narrower width dimension of the nasal bone in the anomaly sample

Motte and Pfister (1960); Linder-Aronson (1970) and Sosa et al (1982) all described typical craniofacial characteristics in "adenoidal facies" children. These included a reduced facial prognathism, a larger anterior facial height and an increased mandibular plane angle. Linder-Aronson attributed many of these differences to a low tongue position, but further research by Solow and Tallgren (1976); Opdebeek et al (1978) and Posnick (1978) suggested that a changed head posture was also involved. Motte and Pfister (1960) and Sosa et al (1982) found craniofacial morphology to be related to radiographic measures of nasopharyngeal airway adequacy. In the present study, with the anomaly subjects exhibiting high nasal resistance coupled with reduced nasopharyngeal and lateral nasal and maxillary dimensions, it was found that the anterior facial height is significantly larger than in the controls, the upper face height n-sp at the $p=0.018$ level and the lower face height sp-gn at the $p=0.019$ level. Other significant differences between the two samples relate to small facial depth, n-s ($p=0.021$), longer clivus length s-ba ($p=0.019$), shorter maxillary length ss-pm ($p=0.005$) found in the anomaly sample as well as the significantly larger ($p<0.001$) variable pgn-cd in the anomaly sample (see Table 40). All these findings support previous research (Wertz 1970; Bushey 1977; Jonas et al 1982; Solow et al 1984). The mandibular angle NSL/MBL was highly significantly larger in the anomaly sample, as was the angle ML/RL ($p<0.001$). These angular values, coupled with the significantly larger linear dimension pgn-cd, suggest that the growth pattern in the anomaly sample has been predominantly downwards, contributing to the long faced

sample compared to the control sample reflect the downward and forward growth of the mandible found in long faced children.

It has been noted in many previous studies of subjects with upper airway problems that the craniofacial morphological differences compared to control subjects included a reduced facial prognathism and a larger mandibular plane angle in relation to the anterior cranial base.

In the present study, the angle pm-s-ba, one of the measurements of nasopharyngeal patency, has a considerably smaller value in the anomaly sample before treatment, having a mean value 3.47° less than in the control sample. However the angle n-s-ba was practically identical in the two samples. Therefore this shows that as the point pm was retruded in the anomaly sample, and the maxillary length sp-pm was identical in the two samples, then the maxillary complex must be retruded in the anomaly sample compared to the control.

The mandibular plane angle NSL/ML also showed a highly significant and larger value in the anomaly sample, as indeed did the angle ML/RL.

Hence the results of the present study support the previous findings, with the maxillary complex in the anomaly sample significantly retruded, both in relation to the anterior cranial base and to the mandible. The anomaly sample also had a larger mandibular plane angulation.

In 1977, Solow and Tallgren showed that extension of the head in relation to the cervical column was associated with a number of craniomorphological factors, findings supported by Opdebeek et al (1978) and Marcotte (1981). In 1970, Linder-Aronson demonstrated that patients with upper airway obstruction due to enlarged adenoids had a significantly different craniofacial morphology from a control group, and in 1979 Solow and Greve demonstrated the association between

craniocervical angulation and respiratory resistance in a group of children before and after adenoidectomy. Their findings showed that a large craniocervical angulation was seen in conjunction with an increased nasal airway resistance in the subjects before adenoidectomy as compared with a control group. Solow et al (1984) reported a significant correlation between the obstructed nasal airway (defined as a small $pm-ad_2$ value and a high NAR), and a large craniocervical angle. The values NSL/CVT and NSL/OPT were used to determine craniocervical angulation.

In the present study, the anomaly sample showed a significant increase in nasal airway resistance but, unlike those with adenoidal problems in previous studies, did not show statistically significant differences in craniocervical angulation when compared to a control group. The reason for this is possibly that in the group that had adenoid problems, the stenosis was in relation to the nasopharynx, hence extension of the head in relation to the cervical column would tend to open the nasopharyngeal airway, making breathing through the nose easier. However in the anomaly group investigated in the present study, the fact that they had bilateral crossbites tended to locate the stenosis anteriorly in the nasal cavity, hence extension of the head in relation to the cervical column would not make any appreciable difference to the passage of air.

The relationship between head posture, craniocervical angulation and craniofacial morphology was the subject of a hypothesis suggested by Solow and Kreiborg (1977). It was suggested that subjects exhibiting increased nasal airway resistance extended the head in relation to the cervical column in order to maintain their vital airway adequacy. This head extension led in turn to stretching of the facial soft tissues, resulting in differential forces on the facial skeleton with consequent effect on craniofacial growth. The net result was the "adenoidal facies".

An analysis of the associations between specific variables relating to anterior facial height and head posture reveals a comprehensive pattern of associations in the anomaly sample (see Table 48). The head posture variables NSL/CVT and NSL/OPT showed positive associations with upper facial height n-sp, lower facial height sp-gn and NL/ML, the angular measurement of lower facial height. Hence a large anterior facial height was seen with a large craniofacial angulation.

Negative associations were seen in the anomaly sample and in the control sample between the head posture variables and the measurements of airway adequacy pm-ad₁ and pm-ad₂. Hence the larger the head posture variables, the smaller the airway dimension value.

It would appear therefore that the present study goes a considerable way to supporting the Solow and Kreiborg (1977) control system hypothesis.

Changes with treatment

Examination of the values of the variables examined after the anomaly sample had undergone treatment with Rapid Maxillary Expansion reveals much to discuss.

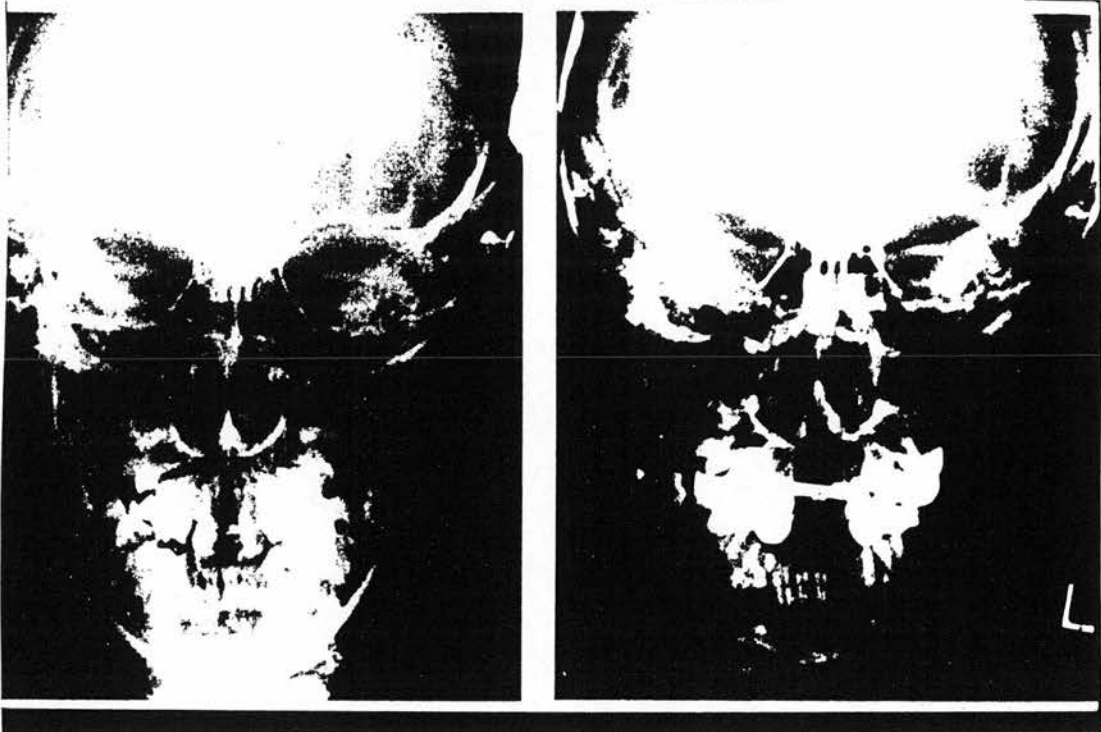
Hershey et al (1976), Turbyfill (1976) and White and Woodside (1989) all reported that nasal airway resistance decreased after an anomaly sample was treated with rapid maxillary expansion. They all found the resistance to have decreased by 40-45%. The present study supports their findings, the anterior inspiratory nasal resistance value decreasing by about 176 pascals/cc/second $\times 10^3$, the expiratory by 145 pascals/cc/second $\times 10^3$, both significant at the $p < 0.001$ level. The total nasal airway resistance also significantly decreased ($p < 0.001$) by 73 pascals/cc/second $\times 10^3$ for inspiration and 84 pascals/cc/second $\times 10^3$ for expiration.

The laminar (k_1) and turbulent (k_2) components also showed a highly significant decreased after RME. These variables have not been studied before in relation to RME, and an interesting pattern has emerged. Warren in 1979 pointed out that

airflow resistance varies inversely as to the fourth power of the radius of the tube through which it passes, so a small increase in the dimension of the tube makes considerable difference to the nasal airway resistance measurement. Certainly it would appear that, within the nose, the area of maximum nasal resistance is anteriorly in the region of the nasal valve and inferior turbinates (Proctor et al 1977; Haight and Cole 1983). Anatomically and radiographically it can be seen (see Figure 46) that the main increase in the width of the nasal cavity following RME is particularly on the anterior nasal floor region (Haas 1961; Wertz 1970; Gray 1975; Montgomery et al 1979; Pavlin et al 1984), so one would expect that there would be a significantly decreased nasal resistance value if, as here in the anomaly sample, the stenosis is primarily anteriorly in the nose. Clinically this is shown by noted ability of the patients to breathe with greater ease through the nose, probably due to the inferior turbinate bones moving away from the nasal septum (see Figure 46B), together with some expansion of the alar valve, leading to less turbulence in the air passing over the inferior turbinates. The observed reduction or complete cessation of snoring, with more restful and productive sleep, is also extremely important.

The Null hypothesis relating to nasal airway resistance are that:

1. There will be no difference in the mean value of laminar and turbulent airflow between the control and anomaly sample
2. There will be no change in the mean value of laminar and turbulent airflow after treatment with Rapid Maxillary Expansion



A

B

Fig. 47 Transverse changes in the maxillary dimensions as a result of treatment with R.M.E.

Of the 36 mean values relating to nasal airway resistance in the present study, 33 showed significant differences, hence it was possible to reject the Null hypotheses for these values. For the remaining 3 values, although they did not reach statistical significance, a definite trend was established.

Overall, therefore, the Null hypotheses relating to differences in nasal airway resistance between the control sample and the anomaly sample, and changes in nasal airway resistance due to treatment can be rejected, in that the results showed that the Nasal Airway Resistance was significantly higher in the anomaly sample, but was significantly reduced by treatment with RME.

The nasal respiratory results of this study support previous research into the interrelationship between RME and NAR, the effects of the reduction in the turbulent component of nasal airflow being of particular interest.

From a clinical point of view, there is an observed change from predominantly oral respiration to nasal respiration after RME.

Utilisation of a cast maxillary expansion splint (see Figure 47) (Grossman 1963) as opposed to a band-borne appliance is of paramount importance in ensuring the rigidity of the expansion, so minimising buccal tooth tip and transferring as much as possible of the expansion force to the midpalatal suture and so to the nasal complex. When the maxillary suture splits it does so in a fan-like manner, wider at the front than at the back of the palate (Timms 1980), and wider at the oral junction of the suture rather than at the nasal site (Haas 1961, 1970). The present study showed significant transverse intranasal width increase after RME, the total width of the anterior nasal base (CNR-CNL) increasing by 10.5%. The changes noted in each half of the nasal transverse dimension, CNR-CNM, and CNM-CNL were approximately the same. This lateral dimensional increase in the anterior part of the nose assumes considerable relevance when coupled with the reduction in nasal airway resistance previously

noted. The relationship between the maxillary suture expansion and upper molar expansion was previously studied by Krebs (1964) using implants. He discovered that the average sutural opening was about one half of the dental arch expansion. Analysis of the mean differences in the present study support this assertion, the intermolar width UMR-UML increasing by a mean of 7.77mm, whilst the maxillary suture width increased by 2.96mm. In addition, the maxillary base EMR-EML only expanded by a mean of 2.15mm, underlying the importance of building in maximum rigidity to appliance design, as even with the cast appliance used in this study there would appear to be have been two components to the expansion, one of the palatal shelf and one of molar buccal tipping. It is the latter that is particularly prone to relapse (Linder-Aronson and Lindgren 1979), and it is possibly true that inadequate band-borne appliances have contributed to the poor relapse record of rapid maxillary expansion. One of the most obvious signs accompanying the opening of the midpalatal suture is the diastema that forms between the upper central incisors. This separation, however, cannot be reliably used as an indicator of suture opening (Haas 1961), and tends to close spontaneously due to the pull of the transeptal fibres, eventually leading to tooth extrusion and arch length shortening (Wertz 1964). This diastema formation was found to be highly significant at the $p < 0.001$ level, the subsequent relapse being prevented by using sectional fixed appliances to the upper incisors, the wire being attached to tubes soldered to the rapid maxillary cast appliance.

The effects of RME on the mandibular arch width dimension were described as unpredictable by Gryson (1977) who recorded no significant width changes. However Haas (1965 and 1970) and Sandstrom et al (1988) noted a significant change after treatment. The present study showed a significant

increase ($p=0.002$) in the mandibular intermolar width after RME, perhaps due to the uprighing of the lower molars as a secondary effect of intercusp activity with the upper molars.

The Null hypotheses to be tested in relation to the transverse craniofacial dimensions were:

- 1) That there would be no difference in the transverse craniofacial dimensions between the control sample and the anomaly sample
- 2) That there would be no change in the transverse craniofacial dimensions of the anomaly sample after treatment with RME.

In turn:

- 1) Control/Anomaly - significant differences were found between seven of the eleven variables examined. Of the remaining four, three - MIR-MIL, MIR-C₂, C₂-MIL - related only to the mesial surfaces of the upper central incisors, so they can be discounted. The fourth variable, that of mandibular intramolar width, did show the anomaly sample mean value to be less than the control, but not significantly so.

The Null hypothesis can therefore be rejected.

- 2) Anomaly sample with treatment - of the eleven variables measured, the mean differences were significant in ten.

The Null hypothesis can here also be rejected.

It can be seen that the effect of RME on the anterior nasal cavity is significant. What then of the changes observed in posterior nasal dimension. The positioning of the maxilla after RME was investigated by Haas (1961) and Wertz (1970). They found that the final position of the maxilla after treatment was somewhat unpredictable, but that there was routinely a downward and forward displacement of the maxilla. Timms (1979) came to a similar

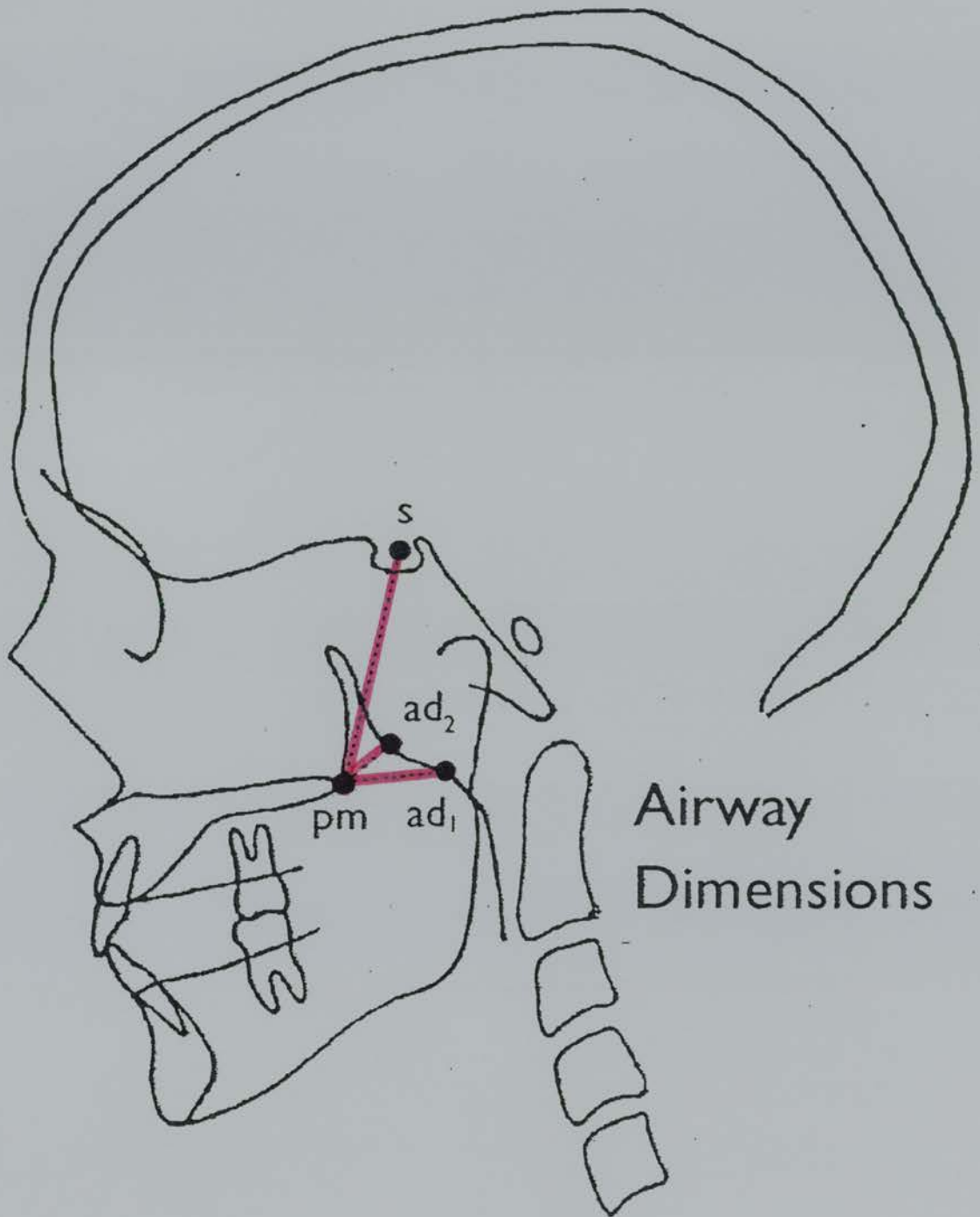


Fig. 48. Significant changes in posterior airway dimensions as a result of treatment with R.M.E.

conclusion, pointing out that the rotation of the maxillary shelves relative to the palatine bone propped the maxilla forwards during expansion. In the present study, examination of the changes in the variables pm-ad₁ and pm-ad₂ due to RME showed that significant ($p < 0.001$) increases in dimension occurred, appearing to support the contention that the maxilla moves forward, hence increasing the size of the available air passageway, and contributing to the decrease in N.A.R. seen after RME. In addition, the linear value s-pm also significantly increased, the maxilla moving apparently downwards as well as forwards (see Figure 48), supporting the findings of previous research. However, there was no change in pm-s-ba so it would appear that the observed airway dimensional changes are largely a result of downward movement of the point pm on the posterior end of the maxilla, rather than forward.

The Null hypotheses relating to airway dimensions were:

- 1) That there will be no difference in the following airway dimensions pm-ad₁, pm-ad₂, pm-ad₃ and tu-ad₃ between the control and anomaly samples
- 2) That there will be no change in the values of pm-ad₁, pm-ad₂, pm-ad₃ and tu-ad₃ as a result of treatment of the anomaly sample with RME.

In turn:

- 1) Control/Anomaly - The Null hypothesis can be rejected for the three variables pm-ad₁, pm-ad₂ and tu-ad₃. However, when pm-ad₃ is examined, the Null hypothesis can be rejected in the male sample only.
- 2) Anomaly with treatment - The Null hypothesis can be rejected with regard to the variables pm-ad₁ and pm-ad₂. However, these are the two variables that are regarded as the standard measures of posterior nasal airway patency.

Given the short duration (mean 3.75 weeks) of the treatment, it should not be expected that linear and angular variables remote from the maxillary/nasal treatment site would change significantly.

After treatment with RME, however, it was found that the anterior facial height (n-gn) increased significantly, especially the lower facial height component sp-gn. This latter dimensional increase presumably reflects the dental change from a bilateral buccal crossbite to amore normal occlusion without buccal crossbites, which occurs as a result of the treatment, hence leading to a propping open of the bite, and the observed increase in the angles NLS/ML, NL/ML and NLS/MBL. The overbite in consequence reduced significantly. The bite opening effect however has been reduced in the present study by using cast capping splints rather than banded palatal expanders, the latter tending to extrude the teeth on expansion (Sarver and Johnston 1989). Bishara et al (1987) found the bite opening that occurs on expansion to be of a temporary nature. Majouran and Nanda (1994) found the use of a chin cap during treatment to be useful.

The linear variables s-pm also showed a significant increase after treatment, this movement having the effect of increasing the dimensions of the nasopharyngeal space pm-ad₁ and pm-ad₂, hence allowing easier passage of air through the nasopharynx.

The Null hypothesis relating to linear craniofacial dimensions were:

1. That there will be no difference in the following linear craniofacial morphology values between the control and anomaly sample

	n-s	sp-pm
	n-sp	ss-pm
	n-gn	pgn-cd
	s-ba	oj
:	s-pm	ob
:	sp-gn	

2) That there will be no change in these values before and after treatment of the anomaly sample with Rapid Maxillary Expansion.

1) Control/Anomaly Sample

Significant statistical differences in both males and females were found in three of the eleven variables tested, pgn-cd, oj and ob. The null hypothesis can be rejected for these variables in both males and females.

In addition, in the males, a further four variables, n-sp, n-gn, s-ba and sp-gn showed significant statistical differences, so the null hypothesis can be rejected for these variables in relation to male subjects only.

In the females two further, different, variables n-s and ss-pm showed significant statistical differences. The hypothesis can therefore be rejected for these variables for female subjects.

2) Anomaly sample before and after treatment

Significant statistical differences in both males and females were found in five of the eleven variables tested: n-sp, n-gn, s-pm, sp-gn and ob.

The Null hypothesis can therefore be rejected for these variables in both males and females.

With the exception of the mildly significant difference in n-s ($p < 0.05$) for males only, the other values showed no significant change. However, as the variables n-s, s-ba, sp-pm and ss-pm are measurements of bone length, one would not expect them to change as a result of a short course of RME.

Taking these constraints into consideration, the hypothesis can be rejected in total.

The Null hypotheses relating to angular craniofacial morphology values were:

- 1) There will be no difference between the control sample and anomaly sample

for the angles	n-s-ba	ss-n-sm
	pm-s-ba	ss-n-pg
	s-n-sp	NSL/NL
	s-n-ss	NSL/ML
	s-n-sm	NL/ML
	s-n-pg	NSL/MBL
		ML/RL

- 2) There will be no change in the above angles before and after treatment with Rapid Maxillary Expansion.

- 1) Control sample and anomaly sample

Of the thirteen variables tested, nine showed significantly different values between the control and anomaly sample. It was thus possible to refute the Null hypothesis relating to these variables.

The variable NSL/NL showed a positive trend, although not statistically significant, and the variables n-s-ba, s-n-sm and s-n-pg were virtually the same in both samples.

- 2) Anomaly sample before and after treatment

Of the thirteen variables tested, nine showed significant differences, and the Null hypothesis can be refuted for these variables.

The other four variables, n-s-ba, pm-s-ba, s-n-sp and ML/RL, remained virtually unchanged before and after treatment with RME.

Solow and Greive (1979) found that the nasal airway resistance and craniocervical angulation was reduced significantly after adenoidectomy, there being a reduction of some 20° in head posture angulation measured two months after the operation, and a reduction of some 30% in nasal resistance. Similar results were also gained by Woodside and Linder-Aronson (1979).

The present study showed no significant changes in head posture as a result of RME, with the exception of the variable OPT/HOR, despite a highly significant drop in nasal airway resistance. The differences in findings between the effects of adenoidectomy and those of RME on head posture may be explained by the fact that in the adenoidectomy anomaly sample the airway stenosis is predominantly in the nasopharynx; in the anomaly sample treated with RME, the problem lies mainly within the anterior part of the nose. In addition, in the present study the short time scale between the recordings (mean 3.75 weeks) may not have allowed sufficient time for long established head posture habits to be corrected.

The Null hypotheses relating to head posture were:

- 1) That there will be no difference in the craniocervical angles NSL/OPT and NSL/VER between the control and anomaly sample;
 - 2) That there will be no change in the craniocervical angles NSL/OPT and NSL/VER in the anomaly sample before and after treatment with RME.
-
- 1) Statistically, it was not possible to refute the Null Hypothesis, but there was a distinct and measurable tendency for there to be a larger craniocervical angle in the anomaly sample before treatment as compared to the controls.
 - 2) There was no significant change here, except for OPT/HOR ($p=0.032$), so it is not possible to refute the Null hypothesis.

Summary

The present study has aimed to develop further understanding of the craniofacial form, nasal airway resistance and head posture firstly in an anomaly sample exhibiting bilateral crossbite, comparing it to a control sample; and secondly to examine the changes that occur in the above parameters when the anomalous subjects are treated with Rapid Palatal Expansion. The control sample was considered representative of the craniofacial form of children in the catchment area and the anomaly sample compared well with those assembled for previous studies. The control sample of 36 children was considered large enough, but problems of acceptance occurred during the measurement of nasal airway resistance, particularly in the posterior part, which reduced the sample size somewhat, especially when sub-divided into males and females for comparison purposes. It is hoped that further developments in rhinomanometrical measurement will obviate this difficulty.

The treatment undertaken on the anomaly subjects followed current best practice and the records that were taken radiographically, photographically and as plaster casts, did not exceed the norm. The short time period between before and after treatment records fulfilled the needs of the study but consideration should be given to a longer term follow-up to investigate the possibility of significant differences occurring over an increased time span in head posture variable changes, and to identify any relapse characteristics. This will be the subject of future work, as will the effect of RME on the soft tissue profile and an investigation into whether the age of the patient within the 10-15 years old window has any bearing on the efficacy of rapid maxillary expansion. However, the combination of a wide array of methodology has allowed certain conclusions to be reached, and has given a new perspective to the complex inter-relationships examined.

7. CONCLUSIONS

A) When the control and anomaly samples are compared:

- (i) The N.A.R. of the anomaly sample is significantly higher, particularly the turbulent (k_2) component.
- (ii) The airway dimensions $pm-ad_1$ and $pm-ad_2$ are significantly narrower in the anomaly sample - mainly due to the position of point pm .
- (iii) The width of the nasal floor $CNR-CNL$, the maxillary base $EMR-EML$ and the upper molar width $UMR-UML$ are significantly narrower in the anomaly sample.
- (iv) The anomaly sample exhibits significantly larger anterior facial height $n-gn$, especially lower facial height $sp-gn$, as well as a significantly longer variable $cd-pgn$. The whole face is thus measurably longer than in the control sample.
- (v) The facial depth of the anomaly sample, measured by $n-s$ and $ss-pm$, is significantly less, and $pm-s-ba$ angle was significantly less, hence the midface is retruded.

The conclusion for this element of the study is that children who exhibit a bilateral dental crossbite with no adenoidal hypertrophy, but with a high nasal airway resistance, show significantly measurable differences in craniofacial morphology, and a tendency to an increased craniocervical angulation when compared to a control group.

B) When the anomaly sample before and after treatment with RME is examined:

- (i) The N.A.R. of the anomaly sample after treatment is significantly reduced.

- (ii) The airway dimensions $pm-ad_1$ and $pm-ad_2$, and the posterior maxillary height $s-pm$, are significantly increased, due primarily to a downward displacement of the point pm . The posterior airway is thus considerably more patent.
- (iii) The width of the nasal floor $CNR-CNL$, the maxillary base $EMR-EML$ and that of the intermolar distance $UMR-UML$, are all significantly increased after RME. Hence the inferior turbinate stricture is alleviated, and the dentition attains normal intercuspatal relationships.
- (iv) Despite the short time interval (3.75 weeks mean) some craniofacial variables show significant changes - the anterior facial height increases as a result of changing lateral intercuspation - this trend has been minimised by using cast splints, and should be a temporary one in the longer term.
- (v) The head posture variables NSL/CVT and NSL/OPT show no significant changes after RME.

Intraorally the appearance and the function of the dentition is dramatically improved.

The conclusions reached in this section of the study are that RME certainly benefits the child in as far as the reduction in nasal airway resistance leads to the alleviation of related apnoeic problems and an improved nasal function, without resort to any surgical intervention intra-orally or intranasally. The main craniofacial changes occur in the increased width of the nasal floor and the maxillary base, together with a downward movement of the posterior part of the maxilla with treatment.

Long term benefits relating to the interception of long face syndrome require further investigation.

8

GLOSSARY OF EQUIPMENT USED

a) Radiography

Cephalometric Radiography Machine

Panelipse with cephalometric attachment

Head Type 100 EC

Anode 3703

Tube E 75 21 N

Date of manufacture 1979

Serial Number 1465

J Morita Corporation
 11-13, 2-Chome Ueno
 Taito-ku
 Tokyo 110, Japan

Parallel Grid

No. 68 02 830 G100E

Serial Number N70 136

Ratio 6:1

Size 24cm x 30cm

70 Absorbing Strips per cm.

Elema-Schonander
 Solna, Sweden

Cassette

Trimax T16 cassette

24cm x 30cm

Rare earth screens

3M Company plc
 Scotland

Radiographic Film

Trimax XD

24cm x 30cm

3M Company plc
Scotland

:

: **b) Rhinomanometry**

Rhinomanometric Machine

NR6 configuration - nasal resistance meter

Zero system - push button

Resistance to Air Flow - approx. 5mm H₂O at 500cm³/sec

Pressure Range - ± 1000 Pa

Flow Range - ± 1000 cm³/sec

Accuracy - $\pm 2\%$

Mercury Electronics
Pollock Castle Estate
Newton Mearns, Glasgow

Computer

British Broadcasting Corporation Microcomputer System

(BBC Model Master B 128)

Acorn Computers
Cambridge

Printer

Epson FX80

Epson UK plc
Dorland House
388 High Road
Wembley, Middlesex

Monitor

Apple Monitor Model AZMZO 10P

Green Phosphor Screen

Serial Number 9952645

Apple Computer

20525 Mariani Avenue

Cupertino

California 95014, USA

Calibration Unit

Dynamic calibration machine

Producing pressure peak at 500 Pascals

Flow peak at 150 litres/sec³

Mercury Electronics

Pollock Castle Estate

Newton Mearns, Glasgow

Nasal Catheter Adhesive Tape

Lenkoflex Tape

Beiersdorf A.G.

Hamburg, Germany

Oral Catheter Tips

4mm Otoscope disposable tips

Keeler Medical Manufacturing
London

Nasal Spray

Otrivine Decongestant

Xylometazoline hydrochloride B.P.

0.1%

Ciba Laboratories

Horsham, West Sussex

c) Digitiser

GTCO Corporation
Digipad 5
Rockville, USA

d) Printer

IBM "Proprinter"

IBM
Livingston, Scotland

APPENDIX

NAME _____	DOB _____	HOSPITAL NO. _____
NO.		<input type="text"/>
AGE		<input type="text"/>
SEX MALE - 1 FEMALE - 2		<input type="text"/>
ANGLES	CLASS I - 1 CLASS II - 2 CLASS II - 3	<input type="text"/>
NASAL INSUFFICIENCY		<input type="text"/>
BEFORE TREATMENT	YES - 1 NO - 2	<input type="text"/>
AFTER TREATMENT	YES - 1 NO - 2 IMPROVED - 3	<input type="text"/>
NOSE/MOUTH BREATHER		<input type="text"/>
BEFORE TREATMENT	NOSE - 1 MOUTH - 2 MIX - 3	<input type="text"/>
AFTER TREATMENT	NOSE - 1 MOUTH - 2 MIX - 3	<input type="text"/>
SNORER		<input type="text"/>
BEFORE TREATMENT		<input type="text"/>
AFTER TREATMENT		<input type="text"/>
PREVIOUS ORTHO.	YES - 1 NO - 2	<input type="text"/>
PREVIOUS SURGERY	NONE - 1 ADENOIDS - 2 TONSILS - 3 NOSE - 4 OTHER - 5	<input type="text"/>
PARENTAL PROBLEM (N)	YES - 1 NO - 2	<input type="text"/>
SIBLING PROBLEM (N)	YES - 1 NO - 2	<input type="text"/>
DATE INITIAL TEST		<input type="text"/>
TESTS YES - 1 NO - 2	X-RAY S/M RHINO PHOTOS	<input type="text"/>
DATE BEGIN ACTIVATION		<input type="text"/>
DATE END ACTIVATION		<input type="text"/>
DATE SECOND TEST		<input type="text"/>
TESTS YES - 1 NO - 2	X-RAY S/M RHINO PHOTOS	<input type="text"/>
DATE THIRD TEST		<input type="text"/>
TESTS YES - 1 NO - 2	X-RAY S/M RHINO PHOTOS	<input type="text"/>
DISPOSAL	EDH - 1 FIFE - 2 OTHERS - 3	<input type="text"/>

Dean of Dental Studies:
PROFESSOR P. SUTCLIFFE
Secretary to the School
of Dental Surgery:



SCHOOL OF DENTAL SURGERY,
OLD HIGH SCHOOL,
12 INFIRMARY STREET,
EDINBURGH EH1 1LT

Tel. 031 667 1011

23 September 1988

Mr. James P. McDonald,
Edinburgh Dental Hospital
Orthodontic Department,
Chambers Street,
Edinburgh, EH1 1JA

Dear Jim,

LOTHIAN AREA DENTAL ETHICAL COMMITTEE

Your request for ethical approval to study 'The Effect of Rapid Maxillary Expansion on Craniofacial Form, Nasal Airway Patency, and Head Posture' has been approved by two members of the Area Dental Ethical Committee. Our normal procedure is that straightforward applications such as the present one are approved by two members of the Committee and their decision is homologated when the whole Committee meets annually. You may therefore proceed with this study when you are ready.

Yours sincerely,

Professor J.C. Southam,
Chairman, L.A.D.E.C.

JCS/mrm

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